

THE GEOGRAPHICAL AND BIOMETEOROLOGICAL CORRELATES
OF CHILDHOOD ASTHMA MORBIDITY IN TASMANIA

by

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STATEMENT OF AUTHOR

Except as stated herein this thesis contains no material which has been accepted for the award of any other degree or diploma in any university. To the best of my knowledge and belief, this thesis contains no copy or paraphrase of material previously published or written by another person, except when due reference is made in the text.

Signed



Graham G. Giles

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ABSTRACT

Environmental aspects of asthma and wheezy breathing have been investigated in three Tasmanian populations; wheezers from the 1968 survey of the 1961 birth cohort, admissions to public hospitals 1972-1977 for asthma and asthmatics from the 1971 birth cohort. Significant spatial clustering was detected in those members of the 1961 cohort defined as suffering from reversible airways obstructions. Geographic concentrations of persons showing respiratory symptoms defined 'good' and 'bad' areas. Such areas differed, indeed were mutually exclusive, for the two sexes. These areas were used as the basis for sampling asthmatics from the 1971 cohort for the purposes of prospective study. Significant clustering was not confined to spatial distributions; hospital admissions over a six year period demonstrated marked temporal variation. The seasonal distribution of attacks differed for males and females and peaks in admissions of either sex were shown to be related to strong weather changes.

A prospective study of wheezy breathing in samples of children from the 1971 birth cohort allowed a closer examination of the interaction between reversible airways obstruction and meteorological variables. Study area selection was guided by the spatial analyses of the wheezers from the 1961 cohort. In each area the children kept daily diaries of their wheeziness for

nineteen months. The aggregated morbidity series for each area were then examined in relation to daily weather parameters and other atmospheric measurements.

Relationships between weather and wheezing were seen to differ between individual times or parts of the series. Major fluctuations in wheeziness were due to the occurrence of certain weather situations. Regression models for each area and sex were constructed to predict wheeziness under various synoptic conditions. With the inclusion of only commonly available weather variables as predictors the level of explanation achieved for certain weather patterns was very high, up to ninety-six per cent. High levels were maintained during tests on new data. During certain weather types, however, wheeziness was low and was poorly predicted by meteorological factors.

Risks to asthmatics differed depending upon sex and were localised both in the domains of space and of time. Maps delineated areas of increased risk and regression equations indicated times of increased risk. Armed with this knowledge, a primary preventive approach could be advocated for the populations at risk.

SECTION 1

INTRODUCTION

1.1 Medical Geography and the Study of Chronic Disease

Introduction

Medical geography is a comparatively new term for an ancient human preoccupation; the interrelationships between environment and health or disease. Much of this holistic, man and environment conceptualisation of health was eclipsed by the post-Pasteurian emphasis of medical science upon pathogenic agents and their control. With the gift of hindsight it is now apparent that the crusades against infectious diseases fought by health services armed with modern medical technology were but a first offensive. The time is now ripe for the transference of interest from disease and its cure to the new battlefield of health and its maintenance.

The health costs of developed countries, for example, are due increasingly to chronic illnesses. These complaints by definition are of comparatively long duration and, in addition, can possess extremely variable natural histories. These diseases are costly from two points of view. First, they are characterised by multiple and extended use of health care facilities and therefore impose both economic and time burdens

upon the health service system. Second, the extended duration of morbidity accompanied by progressive deterioration or intermittent exacerbation of symptoms contributes at least to a sub-optimal quality of life and usually to continued debilitation, incapacity, lifestyle modification, irreversible handicaps and early death.

Biological pathogens are often implicated in the onset or pre-clinical phase of many chronic diseases. They are usually the initial insults or triggers that prepare the way for other opportunistic mechanisms. Chronic diseases are multi-factorial. Usually several insults or triggers will be required to produce an overt disease entity. These contributory factors are often environmental in nature. Factors thought to be related to differences in chronic disease prevalence, incidence, morbidity, and mortality include elements from the biological, physical and psycho-social environments. Some examples are solar and background radiation, climate, hygiene, history of infections, diseases and injuries, occupation, socio-economic status, psychosocial stress and inherited susceptibilities and predispositions. Many of these are unavoidable, for example genetic tendencies, but equally, many can be modified by changes in lifestyle. Chronic diseases, therefore, present a striking example of the need of and opportunity for preventive medicine.¹

Before preventive measures can be applied, the environmental risk factors and their role in the natural history of a given disease must be understood. Health problems are

multi-causal. Realistic explanation can be arrived at only by adopting an ecological, systems-related approach. The environmental and consequently often spatial dimensions of ill-health have begun to stimulate geographers to apply their skills and viewpoint to transdisciplinary health research and the achievement of the overall synthesis. The geographer's view augments the understanding of health problems by adding a spatial perspective to the epidemiological method.

Geography and Chronic Disease

It is not intended here to enter into an extended review or discussion of the historical growth of the sub-field of medical geography. This exercise has been accomplished many times before by leading medical geographers.² Its inclusion in this dissertation would be repetitive. Instead, discussion is to be centred around the contributions of medical geography to the sub-set of chronic diseases. The term "chronic" can apply to almost any disease with an extended natural history be it due to an infectious agent, physical deterioration or psychosocial stress. It is usually applied, however, to a particular subset of physical diseases, for example, arthritis, cancer, chronic bronchitis and emphysema, cardiovascular disease and multiple sclerosis. Their multifactorial nature makes any classificatory grouping very difficult for each of these disease states usually exhibits a history of infection and a strong psychosocial component. An operational definition of chronic disease of this type is virtually impossible but would include

the following elements: variable onset, long-term, degenerative, multi-factorial, and multiple-path.

The contribution of medical geographers to the study of these diseases was assessed by Malcolm Murray in his excellent review.³ He maintained that geographers generally neglected this area of research because of its definitional and aetiological complexity. Any research conducted upon the spatial distributions of these diseases tended not to be concerned with cause and effect but with the allocation of medical facilities and services.⁴

It was Jacques May in his prodigious works on disease ecology⁵ who stressed the importance of the total environment when examining disease distributions. His words are a constant reminder of the complex web of interrelated causes and effects that continually act upon and influence the health of human populations. His mapping efforts encouraged others and for a time medical geographic research was almost solely concerned with the production of atlases,⁶ with cartographic techniques such as cartograms,⁷ and with probability mapping.^{8,9} Mapping remains the initial analysis in most research of this type and often is its sole object.¹⁰ Murray notes that explanation is relatively easy for maps of infectious or acute diseases but not for chronic complaints.¹¹

Both resource-allocation and mapping projects although interesting, valuable and historically important, avoid the major question of how to describe the causal web of chronic disease

and environment interaction using spatial techniques and analysis. One answer is in the detection of space-time clustering.

Burbank's work provided an example of this application in his analysis of cancer morbidity in the United States.¹² His interpretation, however, is limited by the reliability of the biostatistical data he employed. This limitation applies to many other examples of medical-geographic research. Rigorous spatial analysis requires either a total or a randomised, unbiased sample. The customary sources of health data usually satisfy neither of these criteria. Burbank's computer-generated mapping utilised sophisticated techniques but no amount of skill in data manipulation could remove the inherent faults in the original information. However, as McGlashan¹³ rightly points out; health data, especially mortality data, is of a comparatively high and ever-increasing standard when contrasted with morbidity data or with the various forms of social information commonly available at the urban and international levels.

Regardless of their failings, these data can produce meaningful patterns. This is especially true when areal distributions are tested against a theoretical distribution for significant deviations.¹⁴ This probability approach and its modification from that originally postulated by Choynowski, has become an automatic procedure in the initial and exploratory stages of research programmes. Areas of opposite extremes of significance are postulated to illustrate environments either hazardous to or beneficial to health. In these localities the causative or irritant factors are acting most effectively and

ineffectively respectively. This may be due to differences in degree of one factor or to synergisms produced by particular combinations of elements common to certain regions and not to others. Emphasis on the statistical significance of areal differences filters out some of the effects of data errors especially in an area of low population density where the chance occurrence of one or two cases of a disease can give a spuriously high prevalence or incidence rate.

Chronic Morbidity Studies

So far, the discussion has been almost exclusively concerned with mortality data. Mortality, however, represents the end point of a disease process - it is the visible tip of the iceberg of morbidity that lies submerged in the population. Many chronic diseases do not directly kill but slowly debilitate, cripple, modify lifestyles and hence reduce the quality of life of sufferers. Environmental stimuli that manifest, trigger or enhance a given complaint may repeatedly affect/stress a sick person over a lifetime. Repeated insults may have a cumulative effect producing a gradation of severity between individuals of different age, length of complaint or residence. Thus persons suffering from the same complaint but living in different places could have widely differing prognoses.

Morbidity data is subject to arbitrary and differential notification both by medical practitioners and sufferers. The unreliability and poor comparability of official sources of such data, for example hospital admission statistics, has effectively

limited medical-geographic research to designs involving special surveys. For this reason very few studies of chronic disease morbidity exist in the geographic literature.

Anthropogenic, chronic morbidity is usually the result of environmental contamination. Examples range from occupational exposure to carcinogens to breathing polluted air as a side effect of urban living. The dumping of taconite tailing in L. Superior, the Three-Mile-Island incident, the accidental contamination of the State of Michigan with polybrominated biphenyl (P.B.B.) present experiments of opportunity that will all eventually require input from geographers.

Hunter's work on paediatric lead poisoning¹⁵ is a valuable contribution to this research area. His study stresses the importance of factors that mediate the effects of lead contamination. Different populations at risk are defined by proximity to major arterial routes, traffic densities, house-age and paint history, socio-cultural influences upon attitudes toward pica/geophagy and the consumption of canned foods and those physical factors that affect the level of aerosol lead and the level of vitamin D synthesis. Black children living in older housing in the inner cities where kerbside dust can contain 4000-5000 ppm of lead are particularly at risk. This risk is heightened in summer when increased vitamin D synthesis accelerates the rate of lead absorption.

Chronic morbidity of a socio-cultural nature includes a wide range of problems from malnutrition to drug abuse. It concerns the study of man's maladaptation to particular human

environmental situations. Crime, delinquency and deviant behaviour are becoming increasingly fashionable topics of enquiry.^{16,17,18} This type of study is important because the vast and pervasive problems that exist in society are only partly described by mortality statistics. Sakalowsky's study of inebriety¹⁹ probably has more significance than any analysis of the small numbers of deaths ascribed to alcoholic pathologies in Worcester, Massachussets in 1964. Similarly, in most western countries an analysis of malnutrition based on cases of the classical lesions of kwashiorkor, beri beri or rickets would be meaningless because of the small numbers. Following May's²⁰ example in the third world, some nutritional studies^{21,22} are now taking place in developed countries. Here the problem is not under-nutrition, except in poor and otherwise disadvantaged groups, but over-nutrition and poor dietary selection. Ironically, the variety of available foodstuffs in developed countries is very wide. Despite educational efforts, however, the average consumer spends his food-dollar inefficiently. Lacking knowledge of sound nutritional principles and swayed by advertising; economical and nutritious, but time-consuming, foods are rejected in favour of expensive, non-nutritious, convenience foods.

Chronic mental ill-health, again, is usually approached from the aspect of service provision.²³ Smith, however, examines recidivism in mental patients discharged from hospital.²⁴ In this form of non-recuperation that can be broadly interpreted as chronic morbidity, he does not detect any spatial effect. The distance of residence from the facility does not seem to influence

recidivistic behaviour. He notes that some patients may become over-dependent upon community facilities and prefer to transfer their self-responsibilities to the institution. This observation is supported by Davey and Giles²⁵ who interpret a distance decay in first admissions to a mental hospital as over-service of nearby areas. The degree of apparent mental illness in a community can be seen to be partly a product of negative psychosocial forces stemming from the failure of societal institutions to equip people with the necessary skills and coping strategies necessary for living in today's world. Hunter and Brunn's²⁶ discussion of psychosocial stress and mental illness interprets much of the spatial work of Dunham²⁷ and Giggs²⁸ and Levy and Rowitz²⁹ with the "breeder" and "drift" concepts. These assert that socially and economically disadvantaged areas produce more cases of mental illness and that these areas act as a "sink", or end point to which maladaptive individuals eventually migrate and contribute to further environmental degradation.

The study of physical disease morbidity is the least-researched aspect of chronic disease by geographers. This is unfortunate as many chronic physical conditions possess spatial and environmental characteristics that deserve closer examination and could prove fruitful areas of enquiry when viewed from a geographic perspective. The reluctance of geographers to enter this area is understandable. The lack of consistent, unbiased, biostatistical data upon morbidity levels requires special surveys and poses complex definitional and logistical problems.

Some conditions, however, are straightforward. This is particularly true when morbidity is revealed by an easily observed physical sign, for example, hair discolouration, an unusual swelling or a change in skin pigmentation. Schiel and Wepfer's³⁰ work on endemic goitre uses secondary sources to synthesise the historical progress of the disease in the United States. Unfortunately, even with modern iodine prophylaxis, it appears that endemic goitre is increasing in some areas and also occurring in locations where it was previously unknown.³¹ This implies additional and perhaps novel disease pathways. A detailed study of these goitrogenic regions could lead to new aetiological insights and eventual eradication.

Borman's study of rare systemic connective tissue diseases in New Zealand³² concentrated on morbidity statistics rather than mortality for two reasons: the small numbers and poor certification of deaths involved with this disease and the desire to obtain more elegant spatial knowledge than the place of death. The data he used were public hospital records collected from all of the hospitals in a defined study area and then retrospectively re-evaluated by a physician collaborator. Non-definite cases discriminated by a specially-devised scoring system, were excluded from further analysis. Attempts were made to remove the effects of errors and omissions common to hospital morbidity statistics and to confirm residence at time of onset by using electoral records and telephone directories. The analysis was eventually based upon 266 cases detected between 1950 and 1973. These cases covered the entire spectrum of systemic connective

tissue disease. Six constituent diseases were analysed separately. The importance of time of onset was emphasised as it was thought to be the crucial time for any factor from the environment to exert its influence rather than at the time of definite diagnosis. Onset was again interpreted retrospectively from the records by the physician.

Borman used probability mapping to detect significant spatial concentrations in the total morbid populations and the several sub-groups for the three appropriate censuses. Because of the small numbers obtained, only crude non-sex or age specific population incidences were considered. Significant spatial variations in the diseases collectively were demonstrated and were found to be due to significant variation in only two of the conditions; systemic lupus erythematosus and scleroderma.

From an examination of the population structure it was concluded that age at onset generally confirmed the findings of other studies. No urban or rural bias was shown to exist and the small numbers precluded any analysis of occupational differences. An hypothesised relationship between the incidence of systemic lupus erythematosus and sunshine hours was found to be unsupported by the data, though cases of polymyositis and dermatomyositis were significantly concentrated in low sunshine areas.

Morbidity due to Chronic Respiratory Disease

Howe briefly discussed morbidity and absence from work in modern times in the historical context of disease in the United

Kingdom.³³ The complaints responsible for the majority of missed work days and human suffering fell into the chronic physical disease category, for example, bronchitis, arthritis and rheumatism, digestive disorders, hypertension and heart disease. During 1960-1961 in Britain over thirty-five million days of certified incapacity were due to bronchitis and emphysema and another twenty-two billion due to other respiratory diseases. These ailments far outweighed any other single disease or disease category. The wide prevalence of chronic chest complaints and their consequent dominance in morbidity surveys was not restricted to Britain even though bronchitis is known as the 'English disease'. In the NHMRC Australian National Morbidity Survey 1962-1963 nineteen per cent of illness seen in general practice was due to respiratory morbidity compared to twenty-three per cent in a similar British survey.³⁴

Gibson's³⁵ approach to measuring respiratory morbidity was to sample the population of Sydney and to collect spirometry readings. Spirometry measured the health and efficiency of pulmonary function. Variables obtained from spirometry such as vital capacity and forced expiratory volume in half a second were known to be affected by respiratory disease, infections, smoking, atmospheric pollution and a host of other factors such as age, sex and time of day. After standardisation smokers' and non-smokers' scores were analysed separately. When smokers were mapped the whole of Sydney was uniformly below average. When non-smokers were mapped, low scores clustered in areas of increased atmospheric/industrial pollution, illustrating the insidious effects of acceptable exposure levels.

Girt's study of simple chronic bronchitis in Leeds is the most elegant geographical analysis of respiratory morbidity conducted to date.³⁶ He restricted his survey to females over the age of fifteen years in part because of their low occupational exposure, on average, to polluted air. Thirty quadrats of variable size were systematically selected and twenty randomly selected households in each quadrat were approached for interview. Using a standard questionnaire,³⁷ he collected data from the female occupants upon the symptoms of simple chronic bronchitis, smoking habits and occupational and residential histories. Spatial variation in bronchitis was found to be significantly different to that expected by chance when tested against the Poisson distribution.

Two major urban-human ecological parameters that needed to be evaluated in the aetiology of bronchitis were pollution and socioeconomic status. As pollution was known to be related to housing density, it was thought that a positive bronchitis-pollution relationship would be patterned upon the concentric structure of urban residential density. If bronchitis was more closely related to residential status it was considered that it should imitate the sectoral structure of that factor. These two concepts were evaluated by the distance measured from the city centre to the midpoint of each quadrat and the angular measurement between the same two points. Both were found to be significantly related to bronchitis prevalence but the sectoral, socioeconomic differentiation of the city dominated any effect due to the

current pollution. The elevation of the quadrat or the length of residence in any quadrat were also unrelated to the risk of bronchitis. It was concluded that the present levels of pollution were of relatively minor significance to the simple chronic phase of the disease.

Girt went on to use regression and simulation methods to determine the concordance between the levels of bronchitis and smoking habits, past and present living conditions, environmental conditions of past residential areas and occupation. The final regression model combined the effects of smoking and the quality of living conditions both past and present. It seemed that, for women, chronic bronchitis was related to smoking and length of residence in damp, overcrowded housing. The scale of environmental interaction was seen to be at the level of household and lower.

*Biometeorology and Morbidity from Chronic Diseases**

Many chronic physical diseases enjoy a popular reputation for being weather-sensitive. It is commonly held that morbid episodes can be triggered or made worse by certain fluctuations or events in the atmospheric environment. The study of the relationships between weather and climate and disease or morbidity is part of the discipline of medical biometeorology. This sub-field of medical geography also has suffered comparative neglect by geographers.³⁸ The literature on this subject is enormous but very little geographic perspective has been added to the

* A detailed review of biometeorological methods and relevant literature will be given in Section 4; the following serves as a general introduction.

methodologies and practices established by meteorologists, physiologists and physicians.

A standard work in this area is by Tromp.³⁹ Chapter two of his volume describes the biometeorological effect on disease. In addition to discussing the effects of weather and climate on various chronic disease groups, for example, allergic diseases, lung diseases and rheumatic conditions, the beneficial effects of several forms of climatotherapy are also described as well as the variation in pharmacological activity of drugs under different weather conditions. It appears that normal, healthy people demonstrate physiological fluctuations as a result of changes in the physical environment. These stresses usually have no more than a mild stimulating affect upon people but in certain morbid states when an individual is maladapted to the environment and is already under some form of stress the additional stress caused by weather changes can produce an abnormal reaction and heighten morbidity.⁴⁰ The old, the sick and the very young are particularly susceptible to these effects. They may experience an acute episode of sickness, an increase in pain, a lowered resistance or some incapacity or they may die. The reduced seasonality of morbidity in developed nations, for example, has been largely attributed to the increased use and efficiency of household central heating to counteract and diminish the thermal stress of winter.⁴¹

Generally, biometeorological studies of chronic disease morbidity have been non-spatial. If the morbidity history of a defined population can be influenced by the weather in any one

geographic location it is reasonable to assume that a different climate-weather regime will produce a different morbidity history in a similarly defined group. Synchronous studies of meteorotropic responses in populations residing in dissimilar climatic environments should give insights into geographical-environmental effects upon morbidity and hence provide input to preventive measures and the assessment of relative environmental risk. An attempt along these lines is exemplified in McGlashan's work on Burkitt's lymphoma in central Africa.⁴² He found that, contrary to Burkitt's finding, not all lymphomas came from river valleys but a significant number from altitudes above 4000 feet. Any altitude effect, however, was found not to be due to temperature variation but to some other unknown factor that varied with altitude. Singh's⁴³ analysis of the difference in incidence of common diseases at high altitude was more widely-based. He compared 130,000 men stationed at low (<800m) altitudes with 20,000 men stationed at high (>350m <5500m) altitudes between 1965 and 1972. In the men who dwelt at high elevation there was a significantly lowered incidence of diabetes, asthma, hypertension, ischaemic heart disease, rheumatoid arthritis, gastric disorders, skin diseases, psychiatric disorders and anaemia.

Focus of the Research Programme

It was decided to conduct research into the natural history and physical-environment interactions of one chronic respiratory syndrome in Tasmania: the phenomenon of asthma/

wheezy breathing. This complaint was chosen for several reasons both historical and academic. The major historical reason was the Tasmanian Asthma Survey⁴⁴ conducted in 1968. This survey had covered almost every person residing in the state in 1968 who had been born in 1961. Records existed for over eight and a half thousand seven-year-olds. Over seven thousand of these had been resurveyed in 1974. The data set was unique of its kind, in the world and of extremely high-quality. No geographic analysis had been attempted on this data. The Asthma Foundation of Tasmania and the survey organisers graciously gave access to the master computer tapes. The Foundation, furthermore, gave a grant-in-aid to assist the project.

The survey organisers had based their protocol upon symptoms rather than diagnoses. Two symptoms were of primary importance; the history or presence of wheezing and the history or presence of productive cough. Lack of pre-diagnosis controlled inter-observer error and rendered the data set open to future retrospective re-appraisal. Each child was the subject of a clinical examination, including spirometry and a health history questionnaire (see appendix 1). The quality of the data and the ease of access to them and related information made medical-geographical analysis unusually convenient. The achievement of total geographic and population coverage removed the usual sampling problems. All the subjects were of the same age and had had their respiratory health status measured by

standard procedures. The structure of the data allowed a variety of analytical approaches to be undertaken.

The asthma/wheezy breathing syndrome, a chronic disease of imperfectly understood aetiology and natural history, had affected over sixteen per cent of the seven-year-old cohort. Its common occurrence in childhood, its widespread prevalence, its chronic and extended history, its unknown aetiology and its suspected environmental sensitivity made it an ideal subject for geographic and biometeorological analysis. The island state of Tasmania, in addition, provided an excellent epidemiological laboratory of manageable size and with significant variation in microclimate and physical environment.

1.2 The Medical Geography and Ecology of Asthma : A Review

Introduction

Asthma, similarly to epilepsy, was for a long time regarded as a supernatural condition. So much so that a common name for it used to be 'Devil's disease'. Even so, Hippocrates recognised links between its occurrence and physical factors especially winds.⁴⁵ The existing literature upon asthma and its relationship to various factors of the physical and human environments is enormous.⁴⁶ The majority of studies, however, lack comparability and are usually non-quantitative. Also a marked paucity of work exists in the Southern hemisphere. Although a great number of investigations have been accomplished, any synthesis of material is difficult, especially from a comparative point of view. For example, between countries, and within countries between experts, a definition of what comprises asthma differs widely. In addition, the majority of studies describe detailed histories of small numbers of highly selected, and therefore unrepresentative, cases. This, coupled with lack of geographic or socioeconomic sampling allows only very broad general hypotheses to be made in regard to the influence of environment upon asthma morbidity.

Asthma is a general term for a heterogeneous, chronic, respiratory syndrome often found in association with other diseases especially bronchitis. All asthma conditions are characterised by reversible airways obstruction which may or may not be accompanied by wheezing. Severely incapacitating and sometimes

fatal, it is pandemic with a world-wide incidence of about one per cent. The disease is frequently episodic with few clinical or physiological signs of airway obstruction between attacks. It is often difficult for a doctor to tell the difference between asthma, chronic bronchitis, and emphysema. In its early stages it is reversible. Its continued occurrence, however, leads to irreversible changes in the lungs and permanent disability. A hyperactive bronchial constriction mechanism underlies the manifestation of an asthma episode. This reaction can be triggered by a broad spectrum of stimulants that vary from person to person. This sensitivity is caused by the breakdown of mast cells within the lungs releasing spasmogens and inflammatory chemical substances. These quickly produce spasm in the smooth muscle of the lung which contract and narrow the bronchial tubes. In addition, mucous secretion is increased and blocks the airways. Breathing then becomes difficult with wheezing, breathlessness and coughing. Asthma episodes characteristically peak quickly; the sensitivity usually persisting for a few days. Frequent asthma attacks in childhood are a sufficient insult to produce pulmonary lesions and stunt growth. Asthma can at best be viewed as an awkward period of life that has to be lived through. At its worst it involves the acceptance of sub-optimal health and a reduction in both the quality of life and life expectation. Research has shown that sufferers from asthma and allergic diseases generally have a greatly increased chance of developing leukaemia.⁴⁷

Aetiology and Environment

Jacques May in his studies of disease ecology⁴⁸ has always emphasised the importance of looking at the total environment. He considered it to have three major divisions: inorganic, organic and sociocultural. Each category was acknowledged as possessing aetiological elements that could act both independently or synergistically; some effects being additive others multiplicative. His work on malnutrition provided much evidence in support of these views. Nowhere are the relationships between culturally derived values, the ability of the physical environment to produce food and the role of intercurrent infections and parasitic infestations so pronounced as in the manifestation of kwashiorkor or the various avitaminoses that are rife in the Third World. For convenience, May's categories have been adopted here. The complexity of asthma's natural history necessitates some arbitrary organisation of material. These divisions, renamed as physical, biological and human prove to be quite suitable.

The Physical Environment

The elements discussed in this section can all be measured on an interval or ratio scale. They include temperature, humidity, precipitation, wind, ionisation, altitude, coastal proximity and soil type. The ease with which most of these can be measured has encouraged the recording of their variation in time and space. This historical-geographical data has also had the advantage of being easily accessible for use in classifying

physio-climatic regions and for meteorological prediction. This ease of access is responsible, in part, for the enormous literature upon the relationship between particular climatic factors and the prevalence and periodicity of asthma. This wealth of information is, on the whole, of fairly poor quality. Much work lacks reproducibility and little is either objective or quantitative. However, given the highly variable quality of investigation, the consistent findings are remarkable. Some of these are reviewed in this section for their potential as research hypotheses.

Temperature

Asthmatics have been shown to possess an impaired thermoregulatory mechanism. An inability to adjust to sharp changes in temperature, especially cold changes, make them particularly susceptible to the effects of short term variation in atmospheric temperature. The cooling associated with the passage of cold fronts has been suspected for a long time to be responsible for an increased number of attacks.⁵⁰ The inhalation of cold air is known to increase airways resistance; a brisk walk on a cold day could similarly result in bronchospasm. In the longer term the process of acclimatisation may be important. In Europe, asthma shows a seasonal peak in the Autumn.⁵¹ This is probably linked to the first periods of cold weather after the warmth of the Summer. During the extended warm period the asthmatic's thermoregulatory response might adjust to a new temperature/spasm threshold. The onset of the first cold front finds the

system unprepared. Temperature could also have indirect effects upon asthma morbidity. First its obvious covariation with many other weather elements might disguise its true effect. It may act as a surrogate for other elements or complexes. Second, the botanical environment is intimately associated with many physical factors. The flowering of plants and fungal growth, both of which are temperature dependent, produce pollens and spores which irritate the allergic asthmatic.

Humidity

Low humidity is generally beneficial to the respiratory tract as evidenced by the common location of chest sanatoria in deserts and on mountains. Relative humidity usually varies inversely to temperature. Humidity is important to the growth of plants and fungi and also plays a role in the liberation of pollens and spores. It can thus affect the occurrence and concentration of allergens. Fog may be deleterious to asthmatics particularly in association with pollution.⁵² Humidity, like temperature, is a component of meteorological complexes and hence may have statistically significant associations with asthma attacks because of its relationship to other weather parameters. For example, the effect of humidity upon the number and size of ions of different charge is complex and imperfectly understood.⁵³

Precipitation

Rain, snow, hail and sleet have a cleansing effect upon the air. In this way large quantities of pollen, spores and

oxidant gases and industrial particulates are removed from the air thus ameliorating asthmatic health. On the other hand, rainfall, in conjunction with temperature, stimulates the growth of plants and long term relationships might exist between precipitation and allergen abundance. Precipitation is also, of course, related to weather fronts and might, therefore, demonstrate indirect association with asthma morbidity.

Wind

The velocity of the air increases the cooling effect of the atmosphere. This gives rise to the wind chill effect that can exaggerate the temperature drop associated with cold fronts and sea breezes. Wind also acts as a transportation mechanism blowing foreign pollen and spores and pollution from place to place. However, allergen concentrations large enough to precipitate asthma attacks are thought to arise locally as wind generally has too dispersive an effect.⁵⁴ Wind speeds in the upper atmosphere have some responsibility for the propagation of ions by raindrop shearing.⁵⁵

Ionisation

Although the majority of atmospheric ionisation is due to cosmic radiation, local variations in space charge do exist. Thunderstorms, winds (especially of the foehn type), coastal breezes and man-made dielectric fields and air conditioners all manipulate ion concentration and balance. Experimental studies suggest that concentrations of small, positively-charged, ions can induce asthmatic episodes and that negative ions can ameliorate

attacks.⁵⁶ Ionisation is known to vary with altitude, between land and water systems, throughout weather phenomena and between urban and rural settings.⁵⁷ Inhalation of ionised air has been shown to stimulate pronounced physiological disturbances.⁵⁸ These effects are of sufficient strength to make the link between asthma and ionisation plausible as both a direct irritant of the respiratory tract and as an indirect mediator of complex hormone systems. Suspicion has also been directed towards the possible role of ions in the heightened potency of coastal allergens.⁵⁹

Pollution

One of the results of community, especially urban, living is pollution of the physical and biological environment. Asthmatics' respiratory sensitivity renders them especially prone to atmospheric pollution. Oxidant gases such as ozone and the oxides of sulphur and nitrogen increase airways resistance and have an irritant effect on mucous membranes.⁶⁰ These gases are common industrial and domestic wastes in cities. Automobile exhaust also contains quantities of these substances which, under climatic inversion conditions, can become chemically altered and concentrated. The synoptic patterns of weather can, thus, help increase their potency. Heavy rainfall acts in the opposite way, washing them from the air. Any study of the relationship between oxidant gas concentration and asthma episodicity must take these elements into account.

Altitude

Changes in altitude have been approved therapy for asthmatics in Europe for a considerable time. The mechanism is not understood but remission of symptoms is enhanced in many cases.⁶¹ This effect often lasts for some time after returning to the original environment. A common theory is that the concentration of allergens decreases with increasing altitude, the cleaner air obviously being remedial. Why the same remission is often experienced in the reverse situation remains a mystery. A possible related element is change itself, whether in temperature regime, humidity, ionisation, breathing practices or psychological effects.

Coastal Proximity

Similar changes occur when asthmatics living inland go to the coast and vice versa. Again, this could be linked to changes in ionisation, humidity, or sea breezes. Ordman hypothesises a relationship between the humidity and ionisation found in a coastal environment and heightened allergenicity of local house dust. Other coastal effects could be due to the cooling power of sea breezes, to local concentration of aerosols or ozone, or to different pollen and spore flora in coastal areas.

Soils

Investigations in Europe have indicated differences in the prevalence of asthmatics living on different soils. Unfavourable soils are usually those of high clay content and which are poorly

drained. Dampness is thought to promote fungal growth, especially dry rot in wooden houses.⁶³ An allergic type of asthma would be expected in these areas, but the literature contains no reference to rigorous allergy testing by geographic area. Old, wooden houses in low-lying areas are therefore suspect.

The Biological Environment

A great number of asthmatics are atopic: that is, they demonstrate some form of allergic state. This condition may, or may not, be sufficient to produce asthma episodes every time the particular allergen is encountered but, in many cases, allergens are prime sensitisers and consistently produce attacks. Allergies are characterised by sensitivities to organic substances. They are a response of the body's immunity mechanisms made manifest by skin rashes, eczema, hives and asthma episodes. The severity of allergic reaction can be conditioned by emotional factors.⁶⁴ Plants and animals provide the majority of allergens that commonly irritate asthmatics. Animal dander and seasonal foods are known to contribute to attacks but the three most important allergen groups are pollens, spores, and house dust.⁶⁵ Physical factors play a part in the production of these allergens especially humidity and temperature. They may also play a role in the elevation of allergen potency.⁶⁶

Pollens

Pollens have been blamed in cases where there is a Spring peak in incidence of attacks. The pollen grains do not have to

be inhaled to produce a reaction. Swallowed in food, or ruptured on the mucous membranes of the nose and throat, the allergens are quick to enter the bloodstream and produce a response.⁶⁷ Wind dispersed species are the greatest nuisance particularly grasses. However, too strong a wind will dilute the local concentration. Pollens and other allergens from distant sources probably have little effect because of this property of dilution. Some authorities consider pollenetic asthma to be rare in childhood.⁶⁸

Spores

Moulds and fungi need warm and moist conditions for growth and spore release. They are probably most active in places of high humidity and temperature. Large concentrations can be found in damp buildings. These exhibit season variation, being more common in the Autumn.⁶⁹

House Dust

House dust allergy is due to the presence of the house dust mite; a microscopic arthropod that lives on human dander of hair and skin. *Dermatophagoides pteronyssinus* is the common species in Europe and Australia; *D. farinae* in North America.⁷⁰ Mites like warm humid conditions. The greatest numbers are often found in mattresses. Being extremely allergenic, most atopic subjects are allergic to their extract. Their faecal pellets produce the strongest reaction.⁷¹ They probably demonstrate a season fluctuation in population numbers. Coastal conditions have been suggested as most favourable to their growth especially

given the high humidities and temperatures found in these locations.⁷²

Infections

Many theories have been generated upon the effect of chest infections upon the asthmatic.⁷³ Infections, obviously, can increase mucous secretion and produce inflammation. This is often more than sufficient to trigger attacks of wheezing and coughing. Also, such infections could additionally increase sensitivity to other triggers. Alternatively, bacterial and viral insults to the lungs could result in antibody formation and allergic sensitisation.

The Human Environment

Disease patterns can be highly coloured by underlying human complexities. Differences in social class, working conditions, living conditions, dietary habits etc. can be so widely varied as to defy generalisation. Empirical studies of cross-sectional and some selected samples, however, have produced some common factors for exploration. Most of these result from the interactive aspects of family relationships. Genetic variation between individuals with respect to innate susceptibilities and predispositions describes another spectrum of variability that is difficult to assess.

Socioeconomic Status

British studies have shown a greater prevalence of asthma among higher social groups.⁷⁴ Interesting questions arise with

regard to differences between social classes that could preferentially induce asthma. Usually the lower socioeconomic groups have a higher disease prevalence and morbidity. A probable cause may lie somewhere in the process of allergy sensitisation. This may well be explained by differences in infant feeding practice or dietary range.

Infant Feeding

Much concern has been expressed about the demise of breast feeding in western society. The introduction of foreign proteins, in the guise of formulas and cereal products, when the gut lining is at its most permeable and the immune-response mechanisms at their most sensitive, is the probable scenario of sensitisation.⁷⁵ An established allergic condition then chooses a target organ. This is usually the skin but the lungs are often involved.⁷⁶ An initial sensitisation to one substance seems to imbue a sensitivity to a broad band of substances that are potential allergens. The baby that is never breast fed misses the immunity gained from the antibody-rich colostrum of the first few feeds and is, therefore, doubly prone to develop allergy. A larger proportion of asthmatic children are atopic than normal children.⁷⁷ This is often indicated by eczema, especially its flexural form. The question remaining to be answered is the relationship of social class to breast feeding and thus spatial differences in feeding practices. Regional differences in breast feeding have been shown to exist in Tasmania.⁷⁸

Housing

Certain housing sites, materials and fixtures effect colonisation, and therefore possible sensitisation, by moulds and mites.⁷⁹ Situation is important when considering factors such as fumigation and cold-air drainage. Useful comparisons can be drawn by age of building structure, slope, heating system used, ventilation etc. Once again, variation in housing quality will demonstrate a socioeconomic parallel. In this case, however, it is the lower groups that would be expected to be at the greater risk.

Psyche

Asthmatics have been much maligned and stigmatised because of the emotional aspects of the disease. Adult sufferers are often considered to be highly-strung and not quite normal. This often has an effect upon their work situation and employment opportunity. Episodes of asthma can be produced by emotional stress as in other psychosomatic complaints. The actual mechanism by which the attack is induced is probably hyper-ventilation, a common response to stress.⁸⁰ Childhood asthmatics noticeably have attacks due to excitement just before Christmas and other public holidays. Psychotherapy is often successful in alleviating this condition.⁸¹

Much of the literature which exists concerns the asthmatogenic mother, childhood autonomy/opposition and operant conditioning. These three concepts are interrelated. The first theorises that combinations of certain personality traits are

asthmagenic; that is, the mother's behaviour toward her child can promote the occurrence of asthma. Such mothers have been described as three main types: deprived, achievement-oriented and assertive.⁸² The deprived type, most commonly seen as asthmagenic, is anxious, inadequate, is not warm and does not reveal herself or her feelings. The achievement-oriented type has high aspirations, is articulate and again is anxious and defensive. The assertive mother is histrionic and oriented towards impressing and ever-dominating others.

Pathogenic mother-child relationships have also been usefully studied by observing maternal reactions to demonstrations of autonomy and opposition. Observation suggests that, in certain situations asthma may be the cause of ambivalent relationships between the mother and child instead of a result.⁸³ Operant conditioning, conversely, supports asthmagenicity by providing a device. This theory holds that the asthmatic child observes the mother's reaction to asthma episodes. The maternal warmth and attention afforded him when ill is seen as a reward and thus conditions the child to have attacks whenever attention or affection is needed or withheld.⁸⁴

Asthma in Australia

Prevalence and Natural History

Well designed population studies that give accurate and quantitative descriptions of prevalence are very rare in Australia as elsewhere. Most accepted patterns are accrued from physicians' impressions and these are open, obviously, to many types of

subjective error. Geographically speaking, one area of Australia has a considerable reputation for being the asthma "black spot" of the continent. This is a coastal strip in Queensland from Gladstone to Grafton extending some fifty miles inland.⁸⁵ Its bad reputation is based on clinicians' impressions and is reinforced by the considerable asthma research conducted in this area. Until similar state or national investigations have been executed its asthmatogenicity will not have been demonstrated satisfactorily. Climatically, this area is very similar to other internationally renowned, asthma centres such as Durban, South Africa, New Orleans, U.S.A. and parts of Brazil, Israel, Spain and Portugal, each of which has annual mean temperatures of twenty one degrees Celsius and mean relative humidities of about eighty per cent.

Two surveys however, offer some more objective evidence toward estimates of community prevalence. For convenience, these will be referred to as the Melbourne⁸⁶ survey and the Tasmanian survey.⁸⁷ These two pieces of research are similar in many respects but definitional and classificational procedures differ between them thus making comparison difficult. The Melbourne survey obtained a random sample of about four hundred seven-year-old school children from the metropolitan area. The population from which the sample was selected, the seven year age-stratum, demonstrated a prevalence of between eighteen per cent and nineteen per cent. The sample consisted of 315 asthmatic children and eighty two controls. These were to be the subjects of a seven year, longitudinal investigation of asthma's natural

history. Four arbitrary groups: A, B, C and D were defined in the sample ranging from mild, sub-clinical, wheezing to chronic, unremitting asthma. By ten years of age, seventy per cent of the asthmatics experienced remission. About twenty five per cent of the asthmatics continued to have attacks past their fourteenth year. Severe asthma was found to be associated with early onset, usually before three years of age, high attack frequency in initial years of illness, evidence of chest deformity and pulmonary hyperinflation, persisting airways obstruction and growth impairment (weight more than height). This severe group represented a community prevalence of about half of one per cent.

Hay fever, eczema and urticaria were higher in asthma sufferers than in controls. All asthmatic children exhibited at least one allergy when tested with skin-scratches of standard allergen solutions of rye grass, house dust and egg albumen. Severe asthmatics reacted to a wider range of allergens than did the mild asthmatics. Behavioural problems were significantly different from the control only in the most severe cases. Socioeconomic factors were found to be unimportant. Variables assessed included social class, father's occupation, parental age, housing and income. The families of the severely affected children differed only in function. Family stresses, disruptions, resentment between parents and maternal responsibility for economic management were all more common in this group. It was impossible to determine whether this was a sequel to the child's asthma or whether it had existed beforehand.

The Tasmanian survey is more recent and is yet to be analysed completely. The survey examined ninety seven per cent of the seven year old, age-stratum of the 1968 population of Tasmania. This population of 8,410 children born in 1961 was classified into four symptom groups based on the experience of two symptoms: wheezing and productive cough. Group A, 148 children, had experienced wheezing alone (1.8 per cent). Group B, 2,732 children, had a history of cough alone (32.5 per cent). Those 1,210 children who had suffered both symptoms (AB), comprised 14.4 per cent of the cohort. The total wheezing population of 1,358 children (A+AB) was 16.2 per cent. Again atopy was associated with wheezing. No skin tests were performed but hay fever, eczema and urticaria were included on a questionnaire. No socioeconomic or psychological data were collected but the wheezing group had demonstrated more feeding difficulties in the first three years of life. The total wheezy population was generally associated with impairment of ventilatory capacity, atopy and early age of onset.

Seasonal Variation

Asthma morbidity usually demonstrates a bimodal distribution during the year having one peak in the Spring and another in the Autumn. The first is usually attributed to pollen and the second to cold spells. Investigations of seasonality in Australia have tended to use hospital admission statistics rather than representative samples. Crude as this method is, the evidence does indicate a need for continual, clinical

observation of selected groups to elucidate further causal factors. This is especially true when one considers the fact that the admission statistics are neither age nor sex nor disease nor even location specific!

A comparison of admission statistics for the period of 1955 to 1958 in Brisbane, Sydney, Adelaide and Melbourne found peaks in every season though the Spring and Autumn peaks were most significant.⁸⁸ The Spring peak was bimodal in each of the three years examined. A regular sequential, progress was noticed in the appearance of this peak from North to South. The lag between Brisbane and Sydney and between Sydney and Adelaide was about a week. The lag between Adelaide and Melbourne was about two weeks. Arguments were forwarded in support of pollenetic asthma causing the increased number of asthma episodes in the Spring. Evidence against this hypothesis included the fact that the Brisbane Spring peak did not coincide with the month of highest pollen concentration (January), and that pollenosis was an unusual cause of childhood asthma. The latter, viewed in the light of the Melbourne survey, is refuted somewhat by the high percentages of severe asthmatics demonstrating allergy to rye grass (47 per cent of seven year olds and 62 per cent of fourteen year olds).⁸⁹

Summer peaks began at the beginning of February and peaked a month later ending with the end of March. These were associated with early childhood. Infections, especially rubella, were thought to play a large role. The beginning of the school year was thought to assist the spread of infection

through the population and produce the peak towards the end of February.

Autumn peaks were strongest in Brisbane, the intensity of the peak decreasing southwards. Brisbane differed significantly from each other centre at the one per cent level. In each centre the Autumn peak occurred at a time when temperature levels were falling sharply from their Summer levels. This evidence would tend to support theories of asthmatics' inefficient thermoregulation and subsequent poor seasonal acclimatisation. Minor peaks were observed during the Winter in three of the centres, coinciding with peaks in the incidence of infectious diseases.

Brisbane's singularity is thought to be of climatic origin.⁹⁰ Central to the high asthma prevalence strip, it has been much investigated for seasonality and the effect of climate.⁹¹ Autumn and Spring peaks were generally from mid-March to the end of July and from early October until early January respectively. Seasonal variation was seen to diminish with age. Asthma attendances were lowest both with high Summer temperatures, and highest with changing temperatures in Spring and Autumn. Asthma was most prevalent at a mean temperature of twenty one degrees Celsius and decreased as the temperature moved away from this point in either direction. In the twenty six cooler weeks of the year, the hospital attendances showed highly significant, correlations with mean temperature, dew point and three p.m. relative humidity. These were heightened

when a six week lag was used. A lag of sixteen weeks was able to produce significant correlations with precipitation and soil moisture readings.

In the warmer weeks, as temperature, dew point, relative humidity, precipitation and soil moisture increased, the attendances dropped. The peak of the Autumn wave coincided with the steepest drop in weekly temperatures. The increase in asthma after warm, wet weather suggests the growth of allergens, maybe as yet unidentified. The incidence of asthma has been shown to be closely correlated with seasonal flushes in eucalyptus shoot-growth.⁹² This relationship does not appear to be a direct one: the effect could as easily be due to the production of faecal pellets by insects and arthropods feeding on the young shoots. These invisible motes are highly allergenic.

The smooth seasonal waves of morbidity are generally modified by smaller periodicities that introduce fluctuations. Most of the environment elements thought responsible have been commented upon already. Brisbane, again, has been well investigated. Asthma usually decreases during rainy periods and frequently increases during cold-dry changes.⁹³ The North wind in Melbourne is the worst for hay-fever and asthma sufferers. A Southwesterly in Sydney may precipitate outbreaks of allergic rhinitis. In Perth during the Autumn peak of 1968 the daily prevalence of childhood asthma decreased significantly after increases in air ionisation three days previously, increases in barometric pressure two days before and mean temperature one day before.⁹⁴

Some interesting results were obtained from the National Morbidity Survey conducted by the NHMRC between February 1962 and January 1963 in Australia.⁹⁵ This was based upon the attendance records kept over this time period by eighty-five volunteer general practitioners distributed throughout the Commonwealth. No diagnostic criteria were provided. Each physician's diagnoses were taken at face value. This was not without consequence. When paediatric lower-respiratory morbidity was examined bronchitis accounted for 5,105 episodes, influenza 2,188, pneumonia 1,114 and asthma 911. Much of what had been diagnosed as bronchitis was obviously "wheezy bronchitis" or asthma. Furthermore, these children would probably have been treated inappropriately with antibiotics.

Diagnosed asthma demonstrated metropolitan and non-metropolitan differences. The asthma rate per 10,000 episodes of illness in metropolitan areas was ninety five compared to sixty six for other areas. Males presented more frequently than females. For metropolitan areas the ratio was 103:88 and in non-metropolitan areas it was 72:62 for males and females respectively. This urban concentration of asthma morbidity emphasises the relative importance of urban-industrial atmospheric pollution in the morbidity profile of asthma.

An attempt was made to detect varying patterns of illness with varying climatic patterns. Asthma attacks were shown to demonstrate Autumn and Spring peaks (figure 1.2.1). A close association was found between peaks and flowering season when the states were taken separately, for example, the November peak

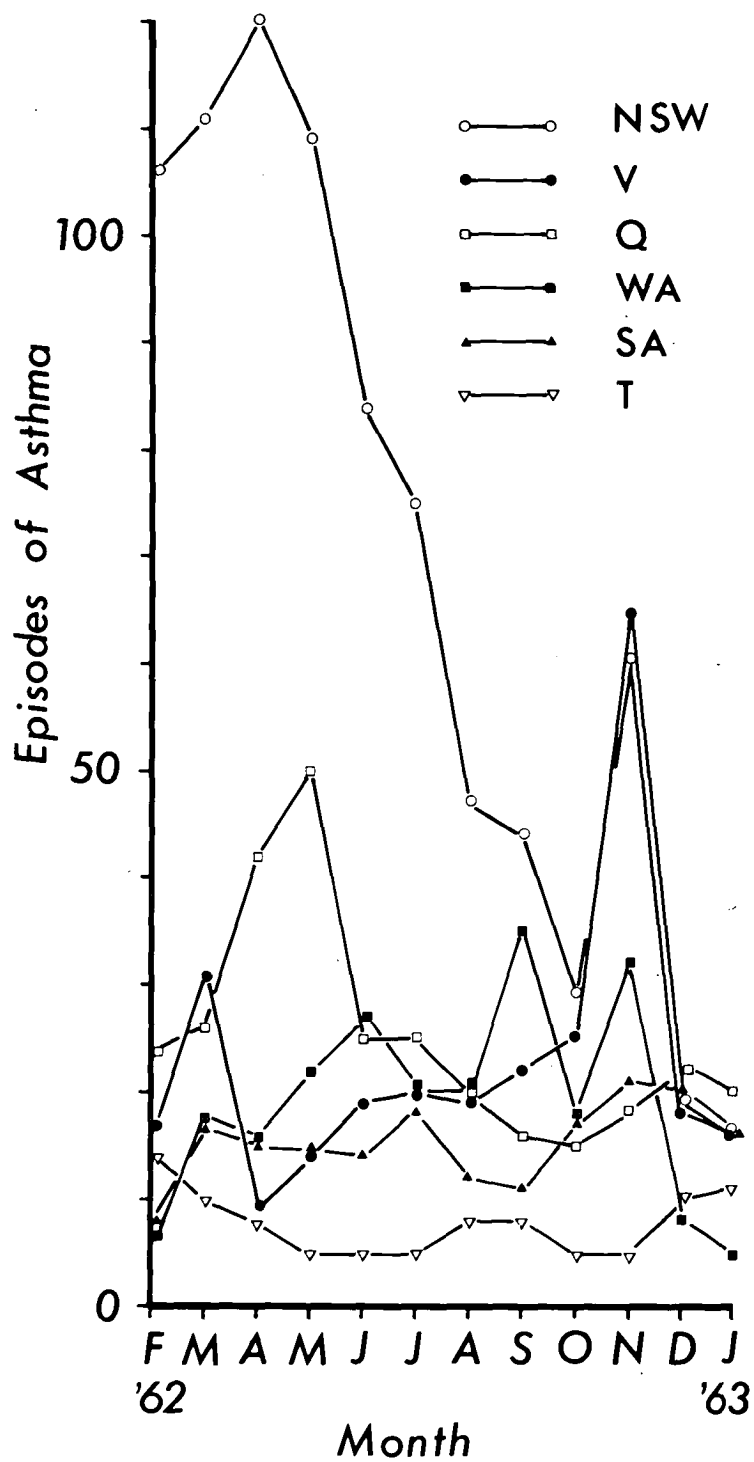


Figure 1.2.1 The Seasonal Distribution of Asthma Morbidity in Australia by State.

(Adapted from NH & MRC National Morbidity Survey, 1962-1963)

in South Australia and Victoria and the earlier peak in Western Australia in September. Profuse Summer rain and consequent growth of grasses and weeds was thought to be linked to the enlarged and extended Autumn peaks in New South Wales and Queensland. House dust sensitivity was thought to play a role in the March peak in Victoria and also in the Autumn peak in New South Wales. House dust collected in Autumn was noted to be more allergenic than at other times of the year. The Tasmanian data presented the flattest seasonal distribution of asthma attacks of all of the states. This was probably due to the smaller numbers involved. The figure was based upon absolute numbers and the states could not have been compared meaningfully until each graph was adjusted to its population base. A false impression, therefore, was given of Tasmanian asthma morbidity.

Asthma in Tasmania

Asthma studies in Tasmania have been few because of the comparatively small population; 402,867 persons in 1976. For this same reason, Tasmania has usually been ignored in national studies of asthma seasonality. In many respects, this is unfortunate as Tasmania provides a unique epidemiological laboratory; an island state with a population small enough not to render longitudinal studies impracticable and a situation where out-migration is low enough to ignore. The entire population can be cross-classified and controlled by virtue of its almost complete isolation from any other.

Investigation of community prevalence has concentrated on populations of school children for reasons of accessibility. The earliest study of this kind had found an asthma prevalence of about eight per cent in primary school children in 1966.⁹⁶ Asthma, in this case, referred solely to physician-diagnosed cases. The later Tasmanian survey, commented upon earlier, gave a prevalence of history of wheezing in seven year old children of 16.2 per cent. About two per cent of seven year old children had experienced wheezing alone while nearly fifteen per cent had demonstrated both wheezing and a productive cough. Boys had a higher prevalence of wheezing to girls in the ratio of three to two.

Upper respiratory symptoms were present in fifteen per cent of subjects with no lower respiratory symptoms (N); thirty eight per cent of subjects who only wheezed (A) or only coughed (B); and sixty per cent of the subject who both coughed and wheezed (AB). In group A the age of onset was younger than in group B, the duration of attacks shorter, and their frequency greater. Group AB tended to be the most severely affected. Group A and AB were similar in their prevalence of general and flexural eczema and food and drug allergies. Unequivocal evidence of airways obstruction was found in the wheezing groups. Hay fever was associated with productive cough.

Mortality due to asthma in Tasmania has been shown to demonstrate late Winter (Spring?), mid-Summer and Autumn peaks.⁹⁸ This varies a little from the Autumn and Spring peaks in morbidity exhibited in studies on the mainland. Mortality also showed a sex

preference, almost twice as many men dying as women. This, again, reflected the innate difference in prevalence between the sexes.

Studies of the natural history of allergens in Tasmania are also rare. Some evidence is available on spores, pollen and the house dust mite. A survey of allergenic mould spores in Hobart during 1960-61 demonstrated that *Cladosporium* was the most common spore followed by *Penicillium*.⁹⁹ It was suggested that these could cause allergic symptoms in the warmer months. *Alternaria* spores were present but not in as significant numbers as in Sydney and were, therefore, thought of as inconsequential. Yeasts were common in winter months but no mention of yeasts was found in the allergy literature.

A flowering calendar for Tasmania, devised for comparison with mainland states, gives the common allergen producers and their season.¹⁰⁰ Common grasses in Tasmania are Bent, Yorkshire Fog, Cocksfoot, Kentucky Blue, Prairie, Rye Grass and Sweet Vernal. These give most problems in November, December and January. Other problem plants are Capeweed (September to October), Broom (October to November) and Plantain (December to January).

The ecology of the house dust mite was the subject of investigation in Hobart in 1974.¹⁰¹ *Dermatophagoides pteronyssinus* was found in sixty one per cent of house dust samples from Hobart suburbs. Densities ranged from 0 to 205 per gram. Their densities in dust from homes of asthmatics were not significantly different to the densities found in dust from homes

of non-asthmatics. Significantly greater densities, however, were found in timber houses. Infestation levels were generally higher than reports from surveys in other countries.

Summary

This literature review described several aetiological relationships based upon mostly European experience and then concentrated on Australian examples. Although Tasmania has been virtually ignored, apart from the 1968 survey, evidence from mainland states would tend to support the need for more rigorous investigations to be conducted here. The general paucity of studies in the Southern hemisphere is an inducement to initiate experiments that can be usefully compared to Northern hemisphere research. Of all the countries and States South of the Equator, Tasmania offers a good epidemiological laboratory. It is isolated by the Bass Strait. The population (400,000) is conveniently reached by modern communication systems. The cohort study conducted in 1968 exemplified this ease of access using, in that case, the School Health Services, infrastructure and personnel.

Asthma has been shown to be an aetiological complex, heterogeneous syndrome. Although much previous work has already been accomplished in Australia, the majority of studies have little explanatory value. The same is true of Northern hemisphere experiments. Criticism can be directed towards quantitative aspects, sample selection and experimental design. The most

popular source of data for longitudinal study has been hospital admission rates. For the most part data were not specific by age, sex, disease nor location. This lack of specificity introduced large errors into spatial and temporal patterns.

In an environmental study of asthma contained within a spatial framework, three elementary precautions have to be satisfied. First, the population samples must be carefully selected to ensure maximal within-group homogeneity. This selection should include some measure of spatial clustering. Second, morbidity monitoring should be on a daily basis and measured on an ordinal if not an interval or ratio scale. Monitoring should be continued for at least one year to take into account seasonal fluctuations. Third, as many aetiological agents as possible should be monitored synchronously with the asthmatics in each spatial unit selected for study.

1.3 Geographical and Biometeorological Studies of Asthma Morbidity in Tasmania

Introduction

The subject of asthma and environment has been a popular area of investigation for centuries. The enormous quantity of research conducted upon asthma, unfortunately, has contributed little to the knowledge of variations from place to place in prevalence or natural history. Definitional and quantitative inelegancies have, in the majority of studies, precluded international or intra-national comparisons or replications. Most of these empirical studies remain location or population specific: their findings cannot be generalised to apply to other times, places or individuals. New work can only view previous findings as potential hypotheses to be tested for validity in different environments. Because of the general definitional and sampling biases inherent in previous asthma studies, the nature, prevalence, natural history and clinical features of asthma remain unclear.¹⁰² In a sense, each new piece of asthma research has had to start from the beginning with a new definition of what comprises asthma.

Although parts of the following analysis, notably sections three and four, will be conducted upon samples of physician-diagnosed asthmatics, asthma will be generally defined here as:

... the history of or presence
of an audible wheeze detected
by the unaided ear.

This definition is given to complement the concept of wheezing as the major diagnostic criterion used in the Tasmanian Asthma Survey¹⁰³ and that of a continuous asthma spectrum proposed by the Melbourne Survey.¹⁰⁴ These surveys' common model assumes that a basic bronchial hyper-reactivity is common to all asthmatics and that hyper-reactivity results in bronchospasm which is manifested by an exhalatory wheeze. The phenomenon of wheezing is taken, therefore, as the discriminatory symptom that separates asthma from other conditions. It appears, from recently published opinions¹⁰⁵ that some individuals with bronchial hyper-reactivity do not wheeze but cough recurrently and respond positively to bronchodilators. But, for the purpose of this research, the wheezy history as an indicator of hyper-reactivity will be maintained as the primary diagnostic symptom rather than bronchial hyper-reactivity itself. The only investigation to have used this criterion was the Tasmanian Asthma Survey.

Two major ideas made this survey unique in the literature. The first was the emphasis upon symptoms; the importance of which has just been described. The second was the total geographic coverage attained by concentrating efforts upon a single year's birth cohort which incidentally controlled for age. The most similar study with this type of geographic coverage would be that of Peckham and Butler¹⁰⁶ who used the results of the National Child Development Study based upon all the children born in one week in March 1958 in England, Scotland and Wales. Any resemblance, however, ended there. The questions asked in the British survey dealt with histories of physician-diagnosed asthma or wheezy

bronchitis. This gave a history of wheezing in eleven-year-old children of 12.3 per cent compared to a history of wheezing in Tasmanian seven-year-olds of 16.2 per cent.

The differences between the two surveys prevent any useful comparisons being made. It is impossible to know if the apparent surfeit of asthmatics in Tasmania compared to Britain is real or definitional. Similarly, many of the asthma-environment studies were based upon such highly-selected and non-randomised samples that the relevance of their findings to other populations is suspect.

Problems and Strategies

The motives of this research are to detect, assess and predict environmental risk to asthmatics living in Tasmania and on this basis to provide preventive information to sufferers and physicians. A recent editorial in the *Lancet*¹⁰⁷ stresses the importance of longitudinal studies particularly for diseases like asthma. It goes on to state that, considering the tendency of bronchial hyper-reactivity to increase with repeated stimulation, every attempt should be made to reduce the number of bronchospasms to a minimum in the hope that the aberrant mechanism will eventually re-set itself. The assessment of environmental risk factors would appear to be an important step towards primary prevention¹⁰⁸ in this context. Before prophylactic action can be effected, however, the vacuum in knowledge of geographic pathogenesis has to be replaced by a comprehensive understanding of both the spatial-temporal variability in asthma morbidity and its underlying provocants.

The geographic distribution of asthmatics, however, defined, has never been rigorously analysed. Occasionally a reference to regional differences will be made in the literature, but this is rare and what is meant by "region" is usually only vaguely described. No geographer has ever attempted to map the disease despite the persuasive encouragement of Sir Dudley Stamp.¹⁰⁹ The first stage in any medical-geographic analysis is to map the cases of disease both in their entirety and in any subvarieties that exist within the diseased population.

The primary objective of mapping is the detection of spatial clustering. Clusters* imply that some kind of epidemic process is effecting the distribution of cases. Their significance can be assessed by probability mapping. Statistically significant areal deviations above the average proportion or norm may represent epidemic foci where causative or irritant factors are acting most strongly. Similarly, areas possessing extremely low prevalences probably represent more salubrious environments where causative agents are either absent or mediated by other elements. In these extreme areas environmental contrast is thought to be at a maximum.

Clusters detected in this way can be used to give a spatial framework to detailed follow-up studies that are designed to test the hypotheses generated by their geographic patterning. For example, if a high proportion of asthmatics were found in a

* Cluster is here defined as any spatial or temporal aggregation of cases that is significantly greater than would be expected to occur solely by chance.

rain forest environment and a low proportion in a dry sclerophyll forest environment it would be suspected that rainfall might be linked to asthma prevalence. A prospective study of sampled asthmatics from both environments could help to determine whether the effect was directly attributable to rainfall or was only indirectly linked perhaps because of different allergens produced in the different localities.

An alternative strategy to the detection of geographic clusters based on the local concentration of morbid cases is to examine morbidity based on environment rather than environment defined by morbidity. In the previous example, that of asthma prevalence and forest environments, the "bad" environment is described by excess cases of morbidity. The opposite point of view is to define the environment on theoretical grounds and then examine the contrasts in morbidity found in the different types of environment. If there are morbidity-environment interactions, it is reasonable to assume that such influences would effect differences in the average morbidity levels from place to place. The direction of effect, increased or decreased morbidity, would separate "good" from "bad" environments.

Unlike the purely spatial approaches, environmental interactions are well documented and, although comparability between different works is low, many of their findings are most suggestive and open to formulation as research hypotheses. The paucity of information concerning asthma prevalence in the spatial domain is balanced by a superfluity of environmental studies in specific locations. Geography and environment, however,

cannot easily be separated for the first describes a dimension in which the other expresses its variation. Environmental differentiation is a geographic phenomenon so for any investigation of environmental variation in health status a spatial framework becomes mandatory.

Environmental parameters tend to belong to two categories of time scale, static and dynamic. By static is meant the comparatively slowly-changing or fixed dimensions of the physical environment such as continentality, altitude, maritime exposure, climate, urbanisation and industrialisation. Dynamic refers instead to the continually varying elements of the environment, for example, levels of pollution, dust, temperature, humidity, wind, and ionisation. In effect, the static parameters are synoptic patterns of their dynamic constituents. Climate, for instance, is described by the long-term average variation of the dynamic components of the weather.

Spatial analysis can relate asthma prevalence to these relatively fixed measures of the physical environment and record, for example, a surfeit of asthmatics at high altitudes. This surplus, however, is not solely explicable in terms of altitude but in terms of the discrete and interrelated elements that vary with changes in that dimension and over the course of time.

After all these considerations, it would seem to be appropriate to investigate morbidity-environment interaction at different spatial and temporal scales. The distinction between debilitative and ameliorative areas can be accomplished by mapping a cross-sectional sample of asthmatics. The extremes of areal

morbidity can then be related to their respective long-term environmental exposures. Any extreme areas possessing sufficiently large population bases can then be sampled and monitored over a period of time in order to elucidate more precisely the mechanism acting between the dynamic elements of the physical environment and fluctuations in morbidity from time to time and from place to place.

Study Overview

Problems of data comparability between previous works and a lack of spatial analysis make it necessary for the geographer to approach certain aspects of the study of asthma *tabula rasa*. However, as the findings from earlier studies point to areas of potentially fruitful research, they cannot be totally rejected. This is thought to be especially true for investigations between morbidity and aspects of the physical environment. In the works reviewed, a high level of consensus is found in regard to the importance of the following phenomena: asthmatics' temperature sensitivity, seasonal fluctuations and the effects of weather changes. In light of this it was decided to limit the study of asthma morbidity-environment interaction in Tasmania to these three elements of the physical environment. On the basis of several considerations which included limitations of finance, resources and personnel, measurement problems and more academically, the lack of documented evidence to support their inclusion; the decision was made to avoid psychosocial and biological factors.

Aims

The general aims of this study were: to examine asthma morbidity in Tasmania by investigating geographic and temporal variations in morbidity levels, to analyse the co-variance of asthma morbidity with elements of the physical environment and to generate models of asthmatics' environmental risk.

Objectives

The objectives were to question theories of asthma prevalence and natural history by testing the validity of certain null hypotheses. There were as follow:

1. *no significant variation exists in the spatial distribution of wheezers or of clinical subsets of asthmatics/wheezers in the state of Tasmania,*
2. *no significant variation exists in the temporal distribution of wheezing episodes in the total population of asthmatics or in subsets of asthmatics defined by clinical symptoms, sex, or residence,*
3. *no significant relationship exists between any wheezy morbidity series and any physical environment series measured over the same time period.*

Structure

The dissertation's structure is based on various types of data used to test these hypotheses. Three sources of data were used: the Tasmanian Asthma Survey data (1968, 1974), hospital morbidity statistics (1972-1977) and specially collected morbidity data (June 1977-December 1978) from sampled asthmatics in selected areas of the state. The Tasmanian Asthma Survey data were used to test the first hypothesis concerned with spatial variation.

The results of these analyses are recorded in section two. Hospital morbidity statistics were used to test the second hypothesis and those elements of the third hypotheses which dealt with temporal variation. These investigations are contained in section three. Selected results of the spatial analysis from section two were used as a spatial framework for section four, which describes a biometeorological case study executed in several locations in Tasmania. Data upon morbidity levels in sample populations of asthmatic children were collected specifically to test for temporal co-variation of morbidity with environmental agents. The final section, section five, attempts to draw together all of the findings into an holistic synthesis of asthma morbidity-environment interaction in Tasmania.

Section 1

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SECTION 2

THE 1961 BIRTH COHORT OF TASMANIA

2.1 Introduction

This section analyses a unique data set of unusually high quality in a variety of ways. It was collected in 1968 when the Asthma Foundation of Tasmania in cooperation with the School Health Services screened all children born in 1961, for upper and lower respiratory tract abnormality. The research design was oriented around symptoms rather than diagnostic entities such as "asthma" or "wheezy bronchitis". This controlled for differential diagnostic errors between observers. Data were collected by two main methods: a health history questionnaire and a clinical examination. (For protocols see appendix 1). Parents successfully completed ninety-seven per cent of the questionnaires and ninety-six per cent of their children were examined by a well-briefed team of school medical officers. Altogether, 8087 cases with both clinical and historical information were obtained. In 1974, 7132 of these children were re-examined (88.2 per cent).

The uniqueness of this data set lies in its almost complete coverage (96.2 per cent), of all people born in a single year

for an entire island state of Australia. The age of the respondents, seven years, also removes the need for adjustments to or considerations of such factors as occupational exposure, or occupational status; history of smoking and, to an extent, migratory habits. Although the cross-sectional survey was executed at a time t certain questions enable some historical reconstruction for times $t-1$ to $t-7$ years; e.g. questions concerning the age of onset and the time elapsed since the last episode. The second survey in 1974 also makes possible an analysis of changes from t to $t+6$ years.

The two key questions centred around the history of wheezy breathing and/or productive cough.¹ Answers to these enabled the categorisation of the subjects into four classes: those who had wheezed only (A) 1.8 per cent, those who had only coughed (B) 32.5 per cent, those who had both coughed and wheezed (AB) 14.4 per cent, and those who had neither wheezed nor coughed (N) 51.3 per cent. Only those subjects with a history of wheezing (A+AB), comprising 16.2 per cent of the cohort, were selected for further analysis. When cases with inconsistent or grossly reduced data were eliminated, 1387 remained. Of these, 840 were boys and 547 were girls. The sex ratio was 153.5 males to every 100 females. The total male and female populations were 4404 and 4146 respectively. The prevalence of male wheezers was therefore 19.1 per cent and female wheezers 13.2 per cent.

Cases were located by the primary school attended in 1968. One of the failings of the survey was that it neglected to ascertain either the exact birth location or the location of

maximum residence during the first seven years of life. In a stable, isolated, population like that of Tasmania, it was assumed that intra-state migration would tend to be low and/or short distance; and that, furthermore, school-age children would tend to inhibit family moves. This was found to be true in a recent survey of the 1971 cohort discussed in section 4; over two years, less than two per cent of the families moved.

All of the analyses conducted upon these data were exploratory; designed to detect significant spatial variations that could be used to formulate environmental hypotheses. First, binary variables were selected one at a time and those individuals possessing that particular attribute were mapped by primary school. As the population of each school was known, the proportion of individuals possessing that characteristic could be calculated. When all the binary variables of interest had been examined for significant geographic clustering, categorical variables were treated in the same way. Individuals were classified into two groups: those who fell into category n of variable x and those who fell into any other category of variable x. Using data from the re-survey in 1974 variations in remissions and new incidence were also assessed in this way.

After examining several individual and paired variables the complexity of the data set became increasingly apparent. The spatial variations of individual variables, although interesting, were often contradictory and confusing. The belief grew that the syndrome of "Wheezy breathing" was not homogeneous; and that within the total population of wheezers existed several sub-groups

with different proportions of various characteristics. To explore this concept three multivariate techniques were used; factor analysis, cluster analysis and discriminant analysis. Factor analysis was used to construct indices of the underlying dimensions of covariance based upon the data's correlation matrix. In this way, considerable data parsimony was achieved in that many variables were reduced to a few operational constructs that accounted for the major proportion of the variance and upon which individuals could be ranked or scaled. Cluster analysis was used to cluster like-individuals into groups while minimising within-group variance and maximising between-group variance. This resulted in a few, major, reproducible classes of asthmatic. The discriminant analysis attempted statistically to discriminate between geographic groups of asthmatics. From selected variables, the group means for each area were forced apart by the construction of discriminant functions. The structure of the functions, like the factor constructs, represented underlying dimensions in the data upon which asthmatics in the different geographic areas could be scaled. Each of the multivariate analyses was followed by an allocation of individuals to groups. The proportion of asthmatics falling into any group was, again, examined for spatial clustering by probability mapping.

This tested the proportion of a given type or degree of asthmatic in the larger population for significant deviation from the state norm by comparing areal variation in proportion to the confidence limits for the norm based upon an appropriate probability distribution. Firstly, individuals of one type were

mapped with respect to their primary school in 1968. After the proportion of that type in the school's population was calculated, high and low prevalence schools were selected by eye and combined with contiguous, neighbouring schools of similar or more extreme prevalence until the proportion started to drop or increase sharply. At this point, the cluster of schools was delineated with a line spaced at mid-distance between the peripheral schools belonging to the cluster and schools external to it. The proportion of that type within the cluster was then tested for significant deviation from the state norm for that type.

Any sub-population prevalence having a proportion greatly in excess of five per cent was tested for significant deviation from the state norm using the binomial distribution of proportions. If the proportion of the sub-population was less than five per cent the Poisson distribution was used instead. The use of the Poisson distribution in this way has been treated elsewhere.² The use of the binomial is very similar. In Tables³ for each total number N in a cluster there are values for any number (n_i) of a sub-group up to $N-1$. If the proportion of the sub-group in the state population is p_i , p_i is the state norm. For each n_i in any N two tolerance ranges are given for p_i ; one at, $p \leq 0.05$, the other for $p \leq 0.01$. If p_i falls within the tolerances' range for any significance level the observed n_i in N differs insignificantly from the State norm. If p_i falls below the tolerance range then n_i in N is significantly greater than the norm at that level and if p_i falls above the tolerance range n_i in N is significantly less than the norm at that probability.

2.2 Binary Variables

Sex

The first variable of this nature to be examined was sex. In the 1387 cases of wheezy breathing, representing 16.2 of the cohort, males were more prevalent than females. In fact, males gave a community prevalence of 19.9 per cent and females 13.2 per cent. Figure 2.2.1 illustrates their respective spatial clusters that reached statistical significance. For the combined sexes (2.2.1.a) only one area stood out; the Northeast was lower than the norm, $p \leq 0.01$. The great majority of the State differed insignificantly from the average. In male wheezers (2.2.1.b) the Northeast quadrant was low, $p \leq 0.05$, and the Huon estuary and Channel area was low at, $p \leq 0.01$. No high prevalence areas were detected. A low prevalence area in the Midlands, $p \leq 0.05$, and a lower area on the Northwest Coast, $p \leq 0.01$, were the only significant locations.

The patterns may have no aetiological significance. Both extremes may be migratory artifacts. The low areas could represent "hostile" environments that asthmatics move out of in order to enjoy a "better" environment or to increase access to health care facilities. Further analyses do not support these explanations, for the basic patterns found in the absolute prevalence of either sex are repeated later in the relative proportion of various types and degrees of asthmatic in the asthmatic rather than the total population. By taking the total asthmatic population as the base from which to calculate proportions in later analyses the spatial variation in absolute prevalence is controlled for. Figure 2.2.1.d

provides an example by examining the distribution of wheezers who had no history of productive cough. This type of wheezer forms eleven per cent of the wheezy population. If the areal prevalence rates are calculated on the basis of the total population, the community prevalence would be about 1.8 per cent. Areas that contain low numbers of wheezers would be expected to have low numbers of this type and vice versa so that maps based on the total community would show, or would be influenced by, the absolute prevalence rates in that community. The proportion of a given type in the asthmatic population is far more revealing because it is independent of the absolute numbers. Hence in figure 2.2.1.d when urban areas are excluded abronchitic wheezers are found to be more prevalent in the North and less in the South, $p \leq 0.01$.

Hayfever

About a third (32.5 per cent) of all asthmatics reported symptoms of hayfever. The major cities were close to or above average but not significantly so. Two low prevalence areas were detectable at, $p \leq 0.01$. These were the Northwest Coast, if the cities of Burnie and Devonport were excluded, and a central portion of the East Coast (Figure 2.2.2.a). The average prevalence in the Northeast was fifty per cent but the low absolute numbers failed to gain statistical significance.

Allergies

Half as many asthmatics reported allergies (14.9 per cent) as reported hayfever. Again, urban areas were close to or higher

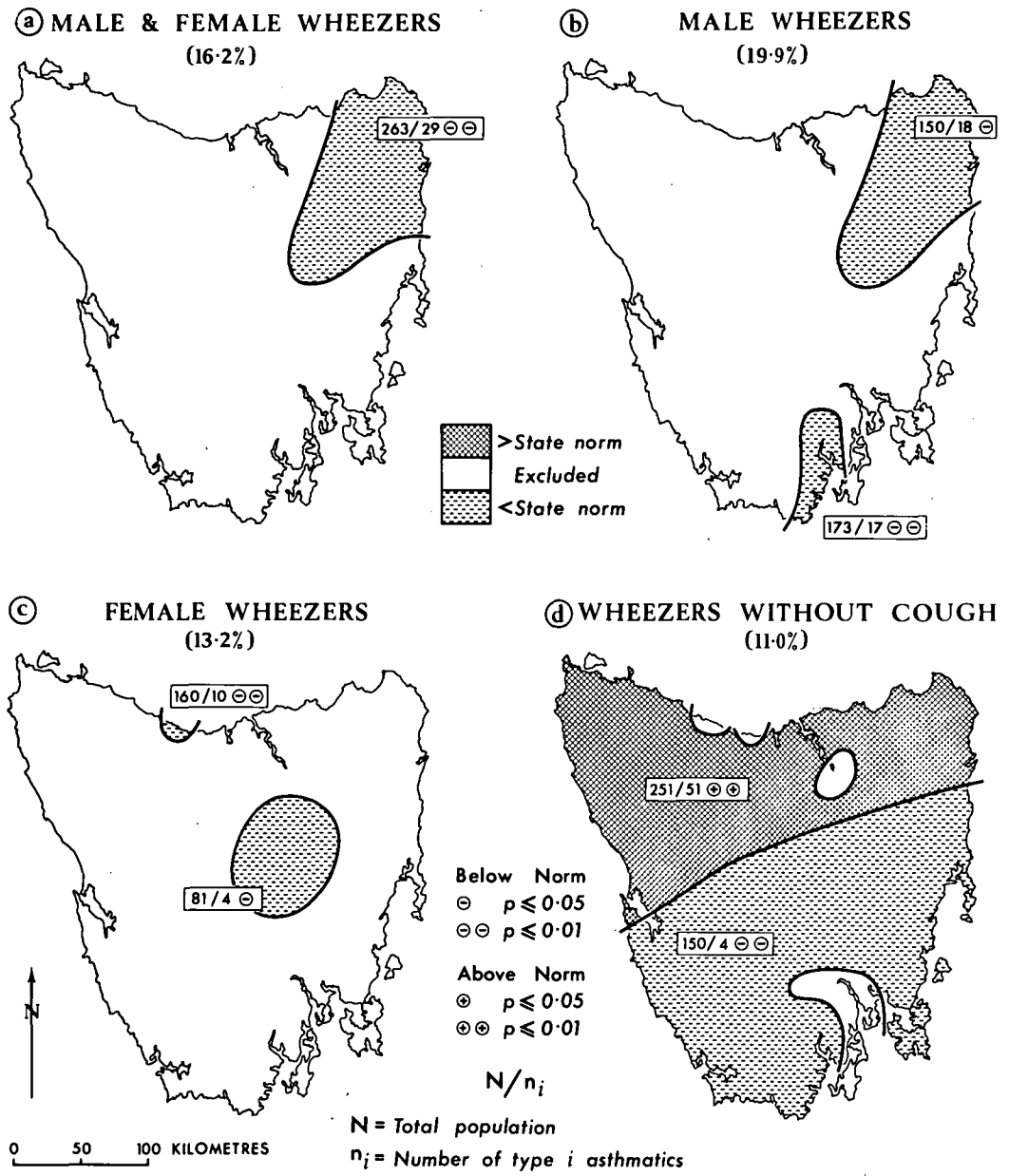


Figure 2.2.1 The Spatial Distribution of Wheezers by Sex

than the norm but did not show any significant deviation. When the Hobart metropolitan area and peripheral settlements and Launceston, the Tamar region and the Northwest Coast were excluded the predominantly rural remainder of the state was significantly below norm, $p \leq 0.01$; averaging about half the normal prevalence, 7.7 per cent (Figure 2.2.2.b).

Pneumonia

A third of the asthmatic population recounted histories of pneumonia and/or pleurisy (Figure 2.2.2.c). The prevalence of these respiratory insults was found to be significantly low in an area that embraced the central highlands of the state and extended northwards to the Devonport area and the North Coast. Devonport's prevalence was twenty per cent in comparison to Launceston's thirty per cent and Hobart's thirty-five per cent. In contrast, the South Midlands and the Southeast quadrant together gave an average prevalence of over fifty per cent but due to the small numbers involved it was only significant at $p \leq 0.05$.

Flexural Eczema

Twenty per cent of the wheezers had evidence of flexural eczema; this was twice the community prevalence. This symptom has long been acknowledged as a sign of atopy and is more noticeable in asthmatics. On the Northwest Coast, in a strip from Ulverstone to Smithton, the prevalence was found to be twenty-eight per cent; significantly greater than the norm at $p \leq 0.01$. South of this coastal strip and extending across the Tamar to the East was a parallel area significantly lower than average $p \leq 0.01$.

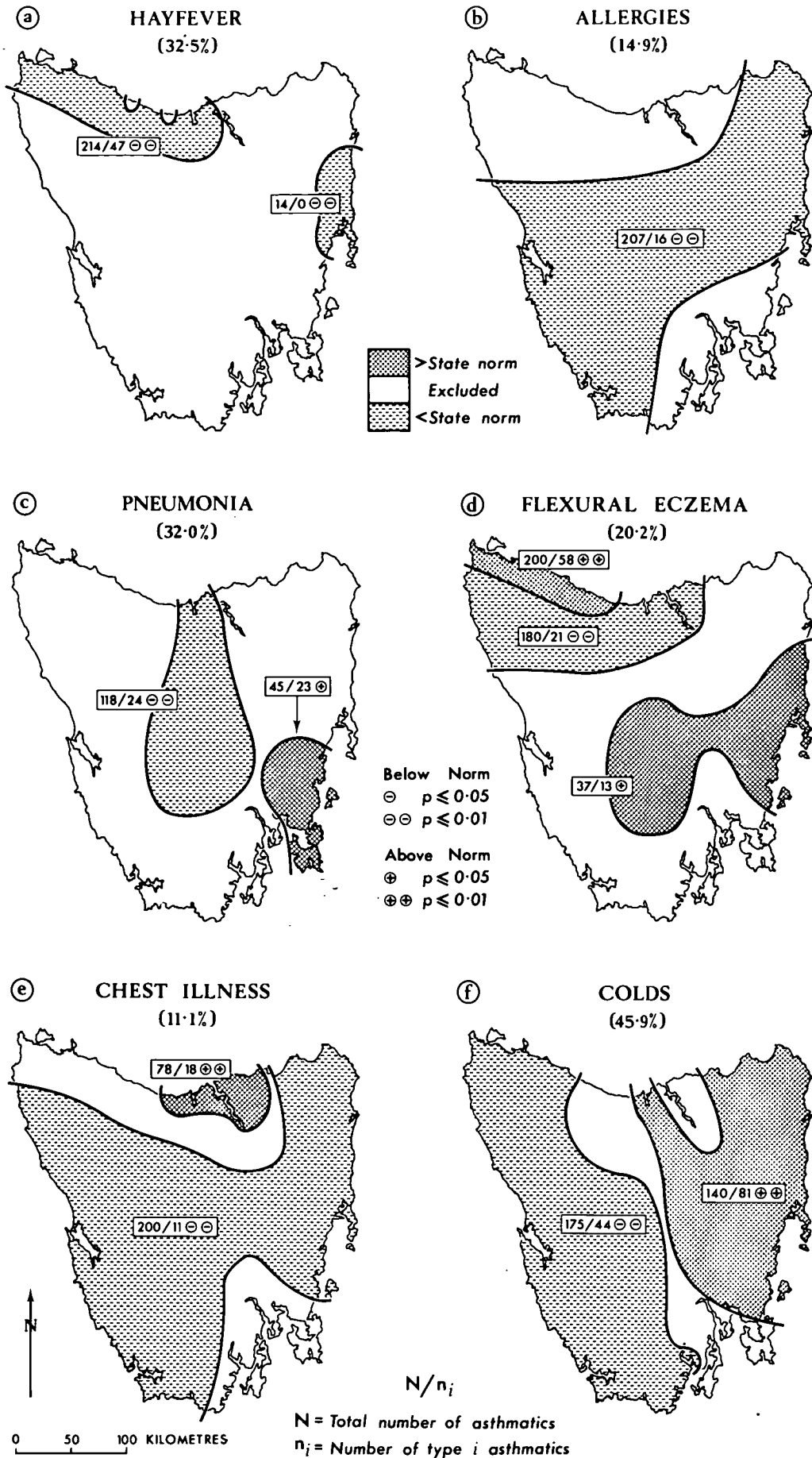


Figure 2.2.2 Signs and Disorders Associated with a History of Wheezing

with a prevalence of only twelve per cent. Another high prevalence area (35 per cent) that included the Central Highlands and the Central East Coast only gained significance at $p \leq 0.05$ due to low numbers (Figure 2.2.2.d).

Chest Illness

Just over eleven per cent of the asthmatics had a month or more of chest illness in the last year. This was particularly prevalent in an area of the central North Coast including Devonport and the Tamar estuary but excluding Launceston (Figure 2.2.2.e). The prevalence in this area was twenty per cent, $p \leq 0.01$, but in Georgetown at the mouth of the Tamar, twenty-five per cent of the asthmatics were affected to this extent. When the Hobart metropolitan area and the Launceston area and the rest of the Northwest Coast were excluded; the prevalence in the rural parts of the State were found to be half of the average, 5.5 per cent, $p \leq 0.01$.

Colds

Over forty-five per cent of the asthmatics said that they had had more than two or three colds a year (Figure 2.2.2.f). When all the urban areas, which were close to average, were excluded a high and a low prevalence area was found. Avoiding the urban areas, the State was bisected longitudinally; to the West the prevalence was low, twenty-five per cent, $p \leq 0.01$, and to the East the prevalence was high, fifty-eight per cent, $p \leq 0.01$.

Summary

The half dozen variables examined so far all demonstrated singular patterns. Geographic variation in prevalence was certainly shown to exist but parallels between variable distributions were not obvious. In the concentrations and deficits that were observed it was difficult to postulate which were due to environmental interaction and which were due to migration. Several patterns did seem to exist that would support hypotheses of rural-urban differences in aetiology.

2.3 Variables Describing the History of Wheezing

A group of five items on the questionnaire measured different aspects of the experience of wheezing. These were related to the age at onset, the time elapsed since the last attack, the frequency of occurrence, the average duration of attack and the total number of attacks. Some of the variables possessed redundant information e.g. the age of onset obviously had a negative relationship to the total number of attacks simply because the older the child was at the onset of wheezing the shorter the amount of time there was in which to have had attacks. The first variables, age at onset and time since last episode, were of special interest because of the underlying time dimension that they measured. Age of onset was used to reconstruct the historical trend in incidence rates; and the time since last episode was used to estimate rates of remission.

Age of Onset

There were eight years of life in which wheezy breathing could have been manifested. These were compressed into four categories for spatial analysis. In the first two years of life thirty-two per cent of the wheezers had had their first attacks. Between the ages of two and three, an additional twenty-five per cent had joined the wheezy population and in the next year another fifteen per cent had suffered their first attacks. Twenty-seven per cent had their first attacks after the age of four. Figure 2.3.1 contains four probability maps; one for each of the age categories. They are based on the proportion of the

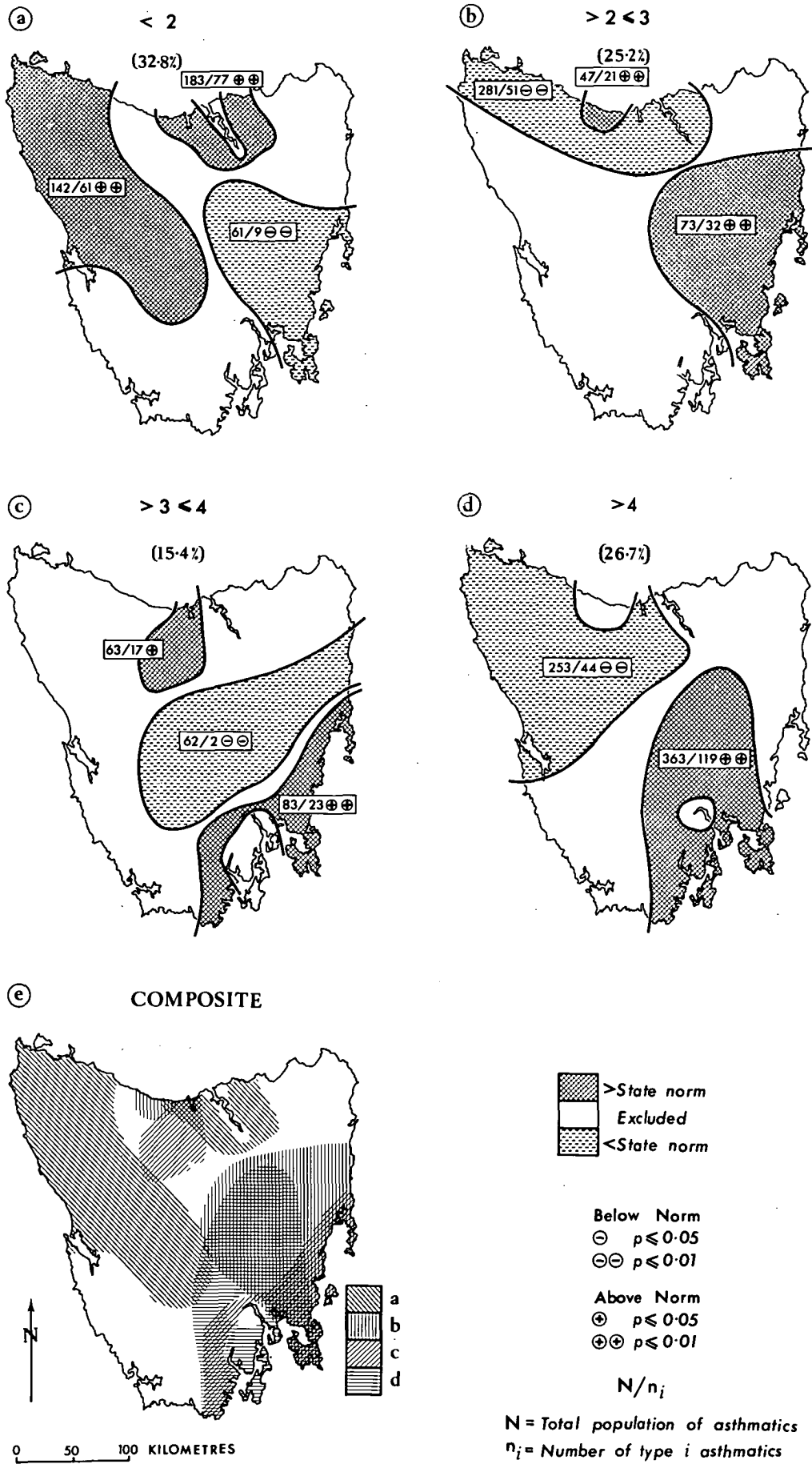


Figure 2.3.1 Age of Onset of Wheezing

total asthmatic population that falls into each category. They show how different areas possess significantly different proportions of asthmatics of a given age of onset. Taken in sequence they demonstrate the changing pattern of incidence over eight years.

Age of onset before two years was proportionately greater, $p \leq 0.01$ in the West-Northwest and in an area peripheral to the Tamar Estuary. It was low, $p \leq 0.01$, in the Southeast. This last area demonstrated a significant excess in the next year's incidence, $p \leq 0.01$. From two to three years the incidence was also high in the urban-industrial strip of the Northwest Coast, $p \leq 0.01$. This area was paralleled by a low-incidence strip that extended inland and to the East and West $p \leq 0.01$. The Southeast, though much reduced in area, remained high for incidence in the third to fourth years, $p \leq 0.01$, matched by an area in the centre of the North Coast, $p \leq 0.05$. These high incidence localities were balanced by a dearth in the interior, $p \leq 0.01$. In the last years the West and Northwest, dominated previously by early onset, were significantly low in incidence of late onset cases, $p \leq 0.01$. The Midlands and Hobart periphery were somewhat higher than the norm, $p \leq 0.05$. Age of onset seemed earliest in the West and extreme Northwest. It took place later on the East Coast and later still in the Southeast and finished in the South and the interior. Figure 2.3.1.e, a composite of figures 2.3.1.a to 2.3.1.d, illustrates this progression.

Time Since Last Episode

This variable was categorised into four time spans.

These were persons experiencing attacks less than one month before the survey (16.5 per cent); those experiencing an episode between one and six months before (34 per cent); those recalling the last episode between six months and two years before (30 per cent) and those who had not had an attack for two years (19.5 per cent).

The last category represents those cases who were enjoying a remission of symptoms at that time. The maps in figure 2.3.2 illustrate these categories similarly to those for age of onset.

The Southeast had a significant surplus of recent episodes, $p \leq 0.01$. This locality extended in area to cover the entire East Coast in the second most recent category. This correlates, to an extent, with the onset maps for this was an area of later onset and one would expect the wheezers in this area to be more active than those areas in which wheezers were prone to earlier onset. For episodes during the last month the rural interior and the West and Northeast were significantly low, $p \leq 0.01$. This area was modified in the second map by the dominance of the East Coast high incidence strip. In both maps the Northwest and urban areas were close to the norm.

Between six months and two years previously, the East Coast changed to a low incidence strip, $p \leq 0.05$, as did the West-Northwest, $p \leq 0.01$; this area extended inland embracing most of the North Midlands. Remissions were significantly high in incidence on the West Coast, in the interior, and in the Northeast, $p \leq 0.01$. The Northwest Coast, the Launceston/Tamar area and most of the Hobart Metropolitan Area were close to the State norm. Low incidences

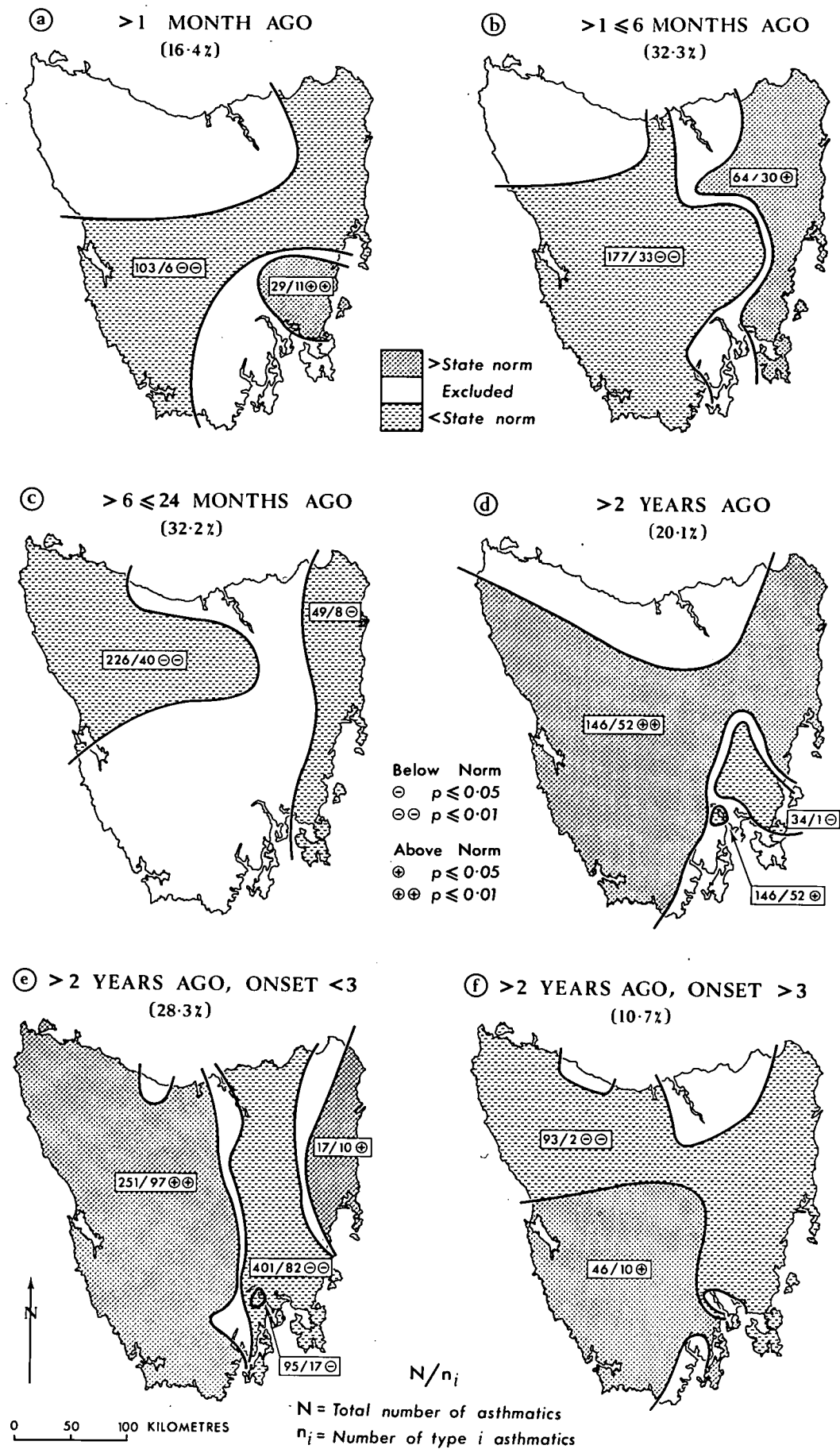


Figure 2.3.22 Time Since the Last Episode of Wheezing

of remissions were not highly significant, $p \leq 0.05$. They were found in the South-Midlands, East Coast and in the Glenorchy area of the Hobart metropolis. A close relationship was apparent between the age of onset and the last episode. This was explored in more detail with respect to remissions.

Figure 2.3.3 graphically demonstrates the percentage distribution of age of onset by year. The column for each year is divided into two parts. The top part is the proportion of those who, in that onset year, were enjoying at least two years' remission of symptoms. This proportion varies from thirty-five per cent to those with onset in the first year of life to none of those who experienced onset in the eighth year. Based on the proportion of remissions, two groups of years can be selected; those with onset under three years and those with onset between three and six years. Those with onset before three years of age enjoyed, on average, 28.7 per cent remissions. Of those with onset between three and six years only 10.7 per cent had a similar status.

All wheezers whose age of onset was less than three years were selected and mapped and the proportion of remissions was again calculated for every school. The results are shown in Figure 2.3.2.e. This should be compared to 2.3.2.d; both portray the proportion of asthmatics enjoying remission of symptoms, 2.3.2.d for the whole population 2.3.2.e for the early onset cases. It is observed that both urban centres, their peripheries and the Midland corridor that joins them, together form a low remission area (20 per cent), $p \leq 0.01$. The East Coast has a

PERCENTAGE DISTRIBUTION OF WHEEZERS BY AGE OF ONSET

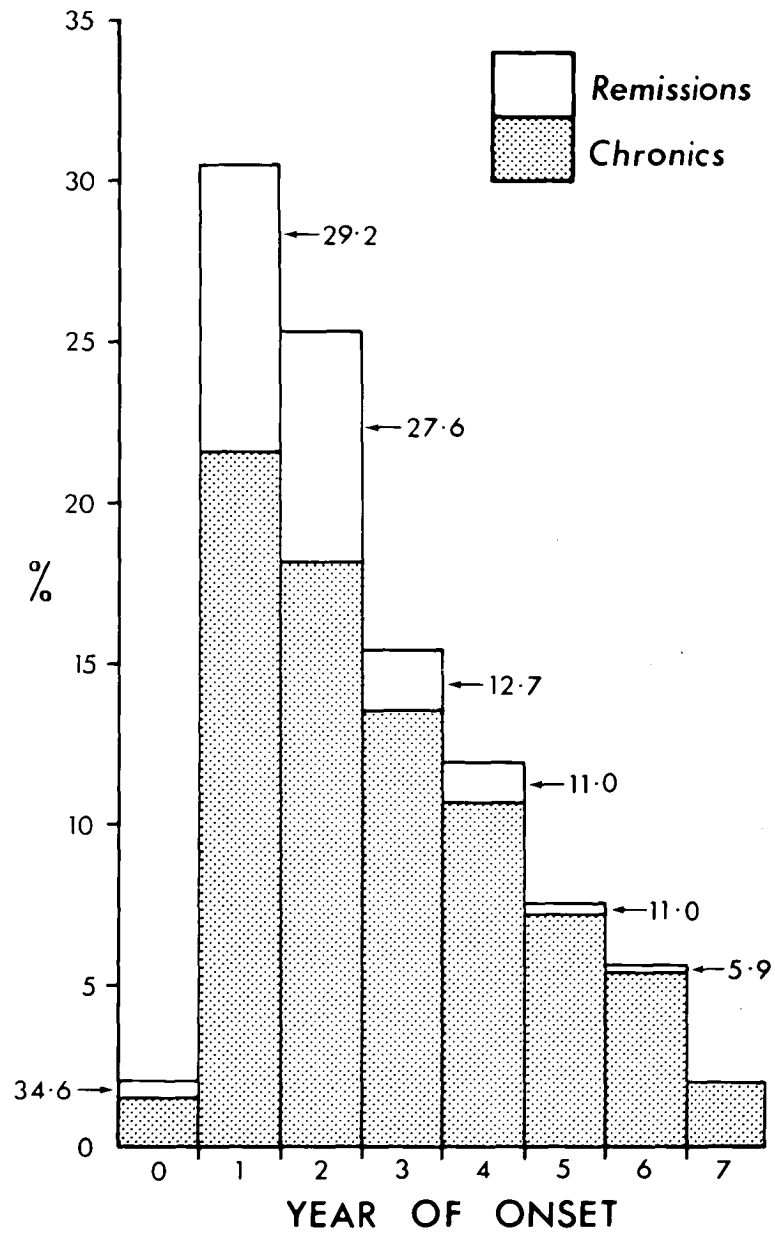


Figure 2.3.3 The Relationship Between Age of Onset and Remission of Symptoms.

surplus of remissions (60 per cent), $p \leq 0.05$, and the Western half of the state a more significant surplus (39 per cent), $p \leq 0.01$.

The patterns were different for late onset wheezers. Only 10.7 per cent of those who had first experienced wheezing between the ages of three and six were enjoying a remission of their symptoms in 1968. The probability map, figure 2.3.2.f, shows a rural deficit of remissions, $p \leq 0.01$, with the exception of the high interior and the Southwest which when combined gave twice the average level, $p \leq 0.05$. The Hobart area, the Launceston area and the Burnie area were all close to average for late onset remissions.

Average Duration of Episode

The majority of episodes tended to be comparatively short in duration. Fourteen per cent were less than twelve hours and fifty per cent were of the length of a day. Nearly two-thirds of attacks were over, on average, in the course of twenty-four hours. A further twenty-seven per cent had attacks that lasted about a week on average. A small proportion, three per cent, had wheezing that persisted for a month or longer.

Attacks of twelve hours or less were significantly low on the Northeast and East Coasts, $p \leq 0.01$, and high in the city of Hobart, $p \leq 0.05$, (Figure 2.3.4). Day-long attacks showed a high incidence, $p \leq 0.05$, in a strip that extended from the South Midland, to the centre of the East Coast. Week-long attacks were prominent along the entire length of the East Coast, $p \leq 0.01$, and correspondingly low in the interior, $p \leq 0.01$. Excluding the Northwest and an area around Hobart, attacks lasting a month or

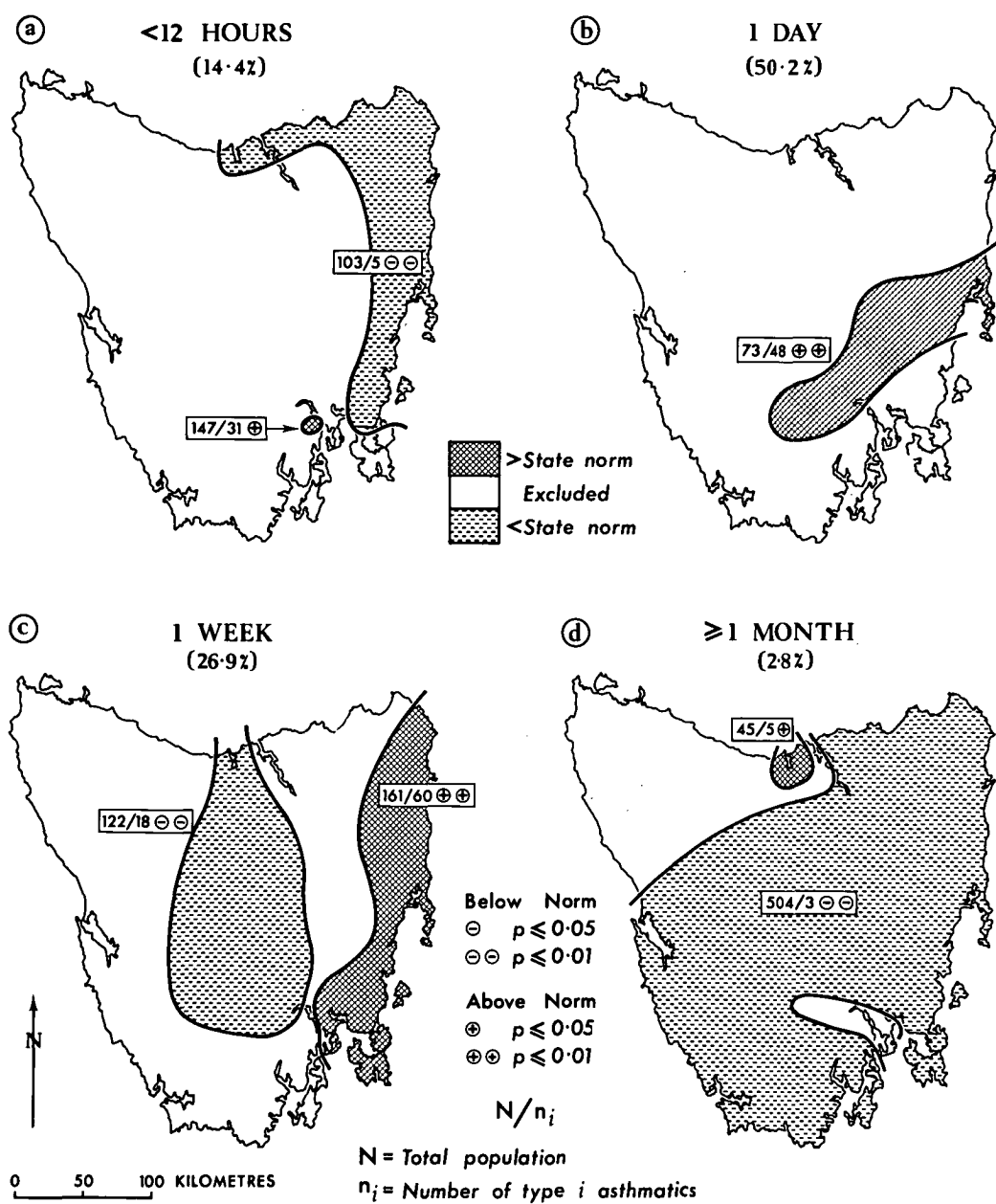


Figure 2.3.4 Average Duration of Episodes of Wheezing

longer were significantly low in the predominantly rural parts of the state, $p \leq 0.01$. An area in the central North Coast was high at $p \leq 0.05$.

Total Number of Episodes

The total number of episodes, another variable influenced by the age at onset, fell into five categories; one attack only, eleven per cent, between one and six attacks, thirty-four per cent, between six and eleven attacks, twenty-three per cent, between eleven and twenty attacks, fifteen per cent, and over twelve attacks, fourteen per cent. The extreme Northwest and the Eastern Shore suburbs of Hobart both had a high incidence of persons with histories of one attack, $p \leq 0.01$. A large rural area was low at $p \leq 0.05$. For one to six attacks there was one low area in the centre of the North Coast. The East Coast had a significant surplus of children with between six and eleven attacks, $p \leq 0.05$. In the two highest categories, eleven to twenty and greater than twenty attacks, no high-incidence areas were found. In both maps the interior and the rural Northeast and the Southwest were significantly low, $p \leq 0.01$.

To control for age of onset, the population was divided into early and late onset groups similarly to the example of remissions using the time of last episode. Early onset cases experienced a seventeen per cent remission rate; late onset a twelve per cent remission rate. Figure 2.3.5.f and 2.3.5.g, respectively, should be compared to figure 2.3.5.e. Early onset cases showed a similar pattern to the total population. The spatial distribution of low incidence moved out of the North East

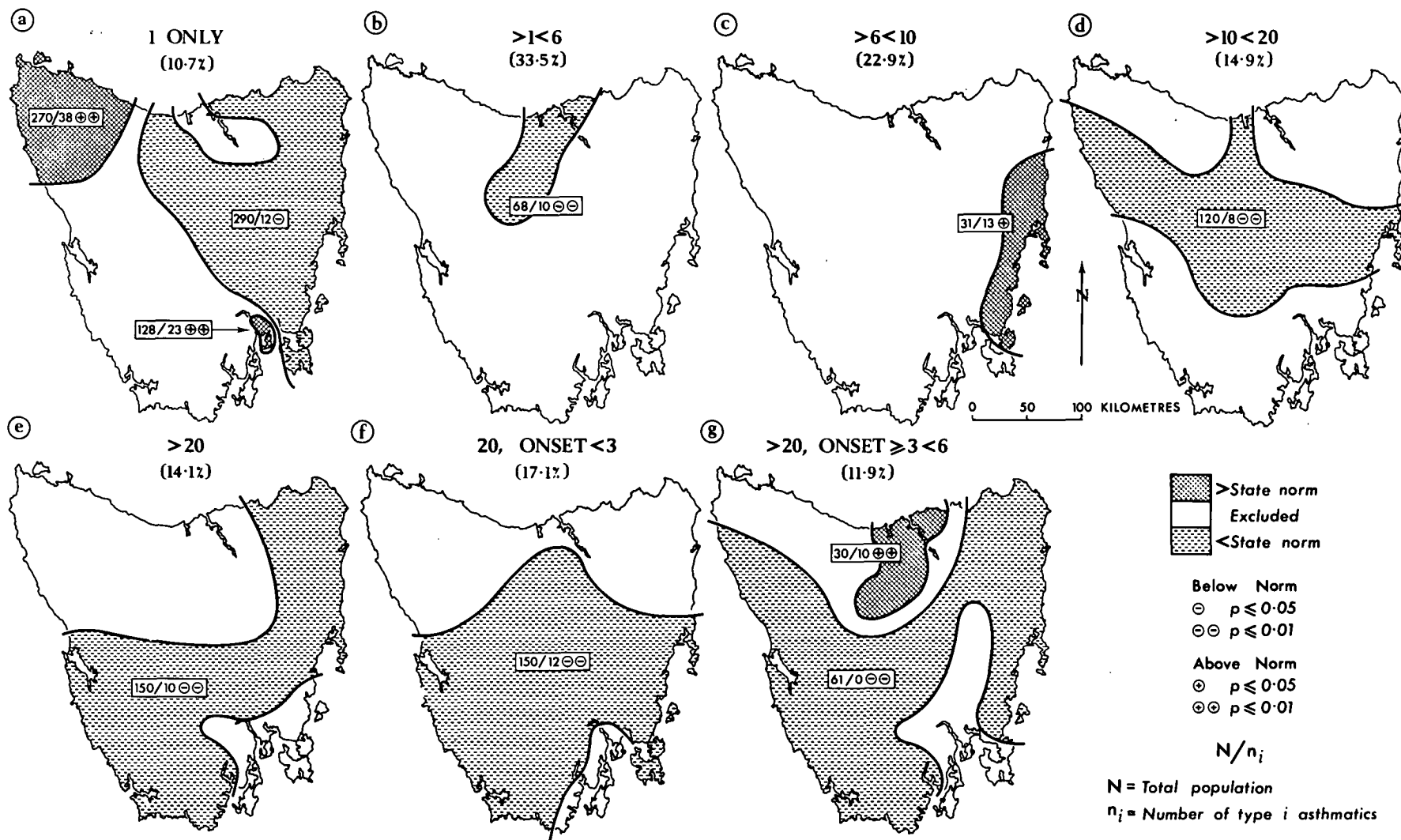


Figure 2.3.5 The Total Number of Episodes of Wheezing

and instead covered the North Midlands. Late onset cases who had experienced twenty attacks seemed to be a composite of figures 2.3.5.b and 2.3.5.e, a rural low incidence coupled with a high incidence in the central Northern part of the State.

Periodicity of Episodes

On average, nineteen per cent of the wheezers had had at least one episode a month, thirty-six per cent had had between one and four a year and twenty-one per cent had only had one attack a year. In addition, 19.8 per cent claimed not to have had an attack in the last two years. This closely matched the proportion of those who answered the question on time since last episode as greater than two years (19.5 per cent). Persons averaging at least one attack a month were high in incidence on the central North Coast and peripheral to Hobart and the Midlands, $p \leq 0.01$. The Northeast was low for this category, $p \leq 0.05$. Up to four attacks a year had been experienced by a significant proportion in a large rural area that embraced the entire East Coast, North Midlands and the upper West Coast, $p \leq 0.01$. Annual attacks were significantly low in the Western half of the state, $p \leq 0.01$. The pattern of remission was not identical to that obtained from the last episode information. The Northeast was high, $p \leq 0.05$, at the same level as the West Coast and the New Norfolk area. A low incidence area, $p \leq 0.05$, was found at the mouth of the Tamar.

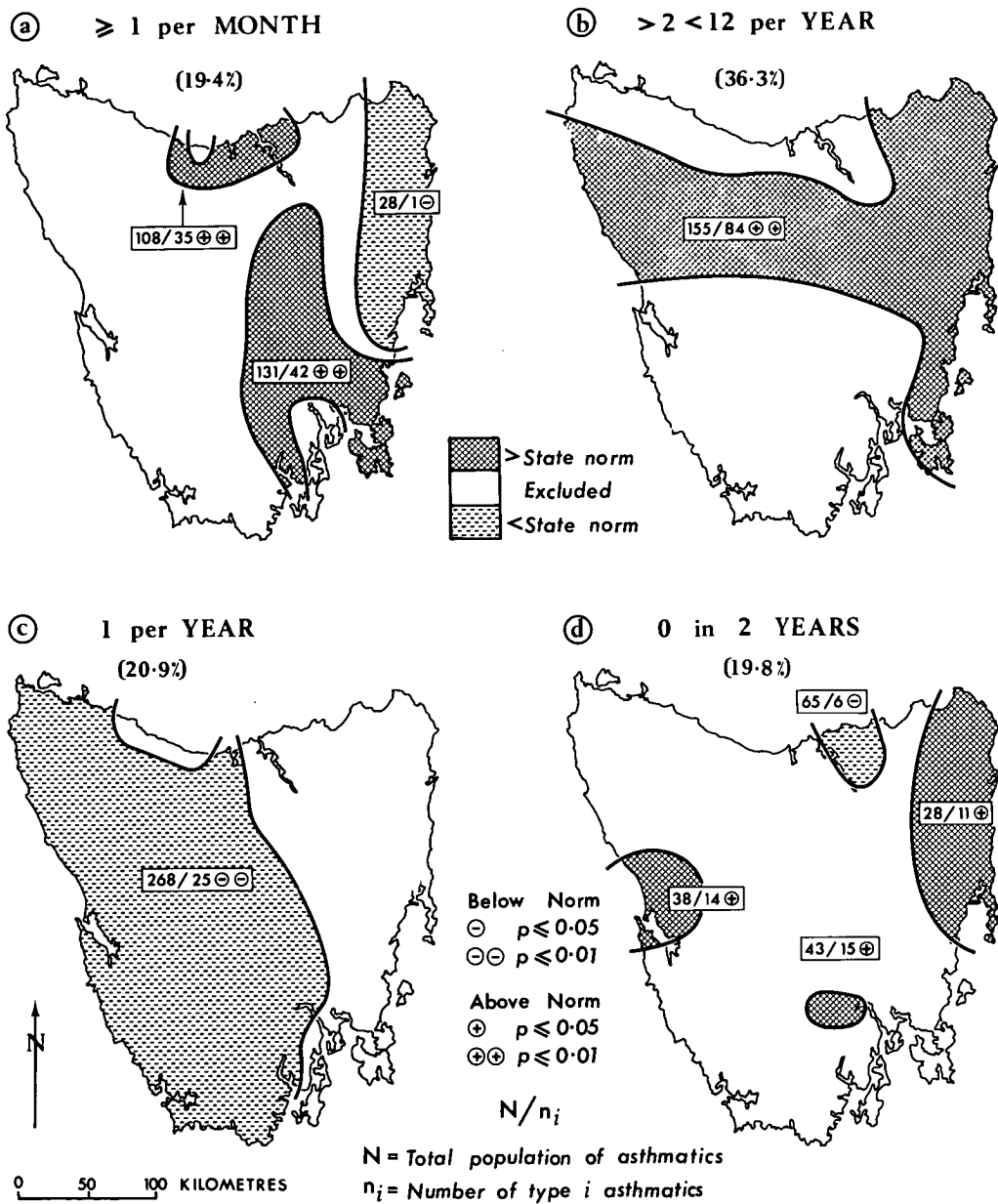


Figure 2.3.6 The Periodicity of Episodes of Wheezing

2.4 Variables that described the History of Productive Cough

The questions used to obtain measures of wheezing history were repeated for the history of productive cough. Productive cough was defined as any cough that was "loose" whether or not sputum was produced. Eighty-nine per cent of the wheezy population reported at least one episode of this chest condition. Because the proportion of non coughers varied from North to South, probability mapping for the cough variables was based upon the coughing population only. Although maps were produced for every variable, only three are illustrated here; the age of onset, the time since the last episode and the total number of attacks. The frequency distributions of the other two variables; duration of attack and frequency of attack were extremely leptokurtic. The distribution of cases into categories was so narrow that it made an analysis of variation in proportions useless. This was a direct result of pre-coding the survey questionnaire's responses.

Age of Onset

The pattern of age of onset of productive cough showed little resemblance to those for wheezing (Figure 2.4.1). Onset under the age of two was concentrated in the Tamar estuary, $p \leq 0.05$ and lacking in the Huon estuary, $p \leq 0.05$. New incidence during the third year appeared in the Southeast and was low in the Northeast, $p \leq 0.05$. During the fourth year, there was a deficit of new coughers in the Northwest when the local urban centres were excluded, $p \leq 0.01$. The central East Coast gained a significant surplus in the same period, $p \leq 0.01$. Late onset was characterised

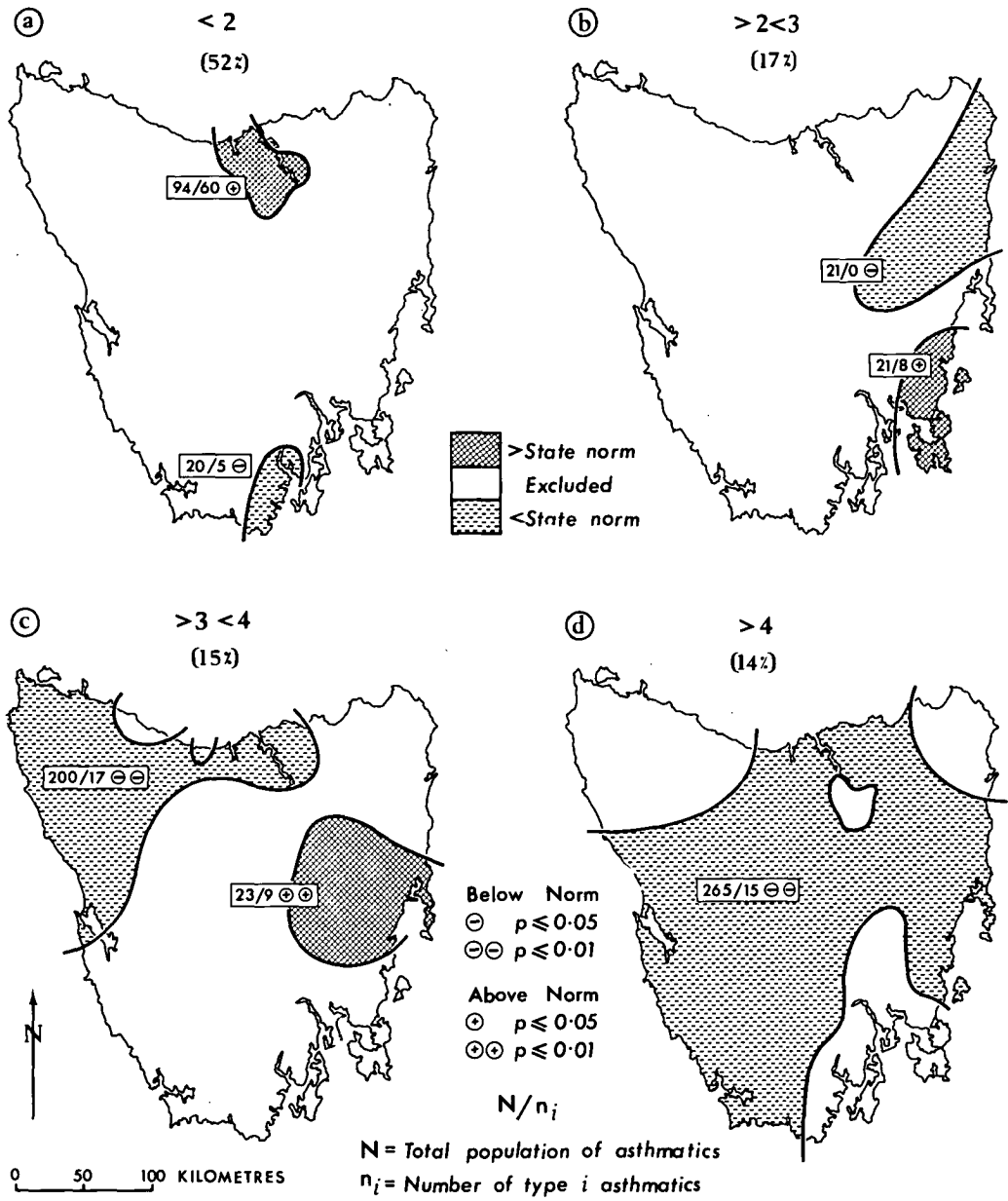


Figure 2.4.1 Age of Onset of Productive Cough

by low incidence in the majority of the rural parts of the State and average incidence in the two urban centres and the Northeast and Northwest extremities.

Time since the Last Episode

Persons having had attacks less than a month ago formed a band of high incidence that stretched from the Northwest Coast to the Southeast (Figure 2.4.2). Attacks from one to three months before were concentrated in the Northeast and were low in the centre of the East Coast. The map of the incidence of attacks between three and six months previously was very interesting; a large rural area was significantly high, $p \leq 0.01$, yet a strip behind the Northwest Coast, inland and predominantly rural, was low, $p \leq 0.01$. Between six and twelve months before the survey a very localised concentration of attacks took place in the Northern and East Shore suburbs of Hobart. Wheezers free of cough for over a year were under-represented in most rural parts of the State but remissions were high in the Northeast, $p \leq 0.01$. The Northeast was also high for remission of wheezy symptoms (Figure 2.3.2.d) but in this case was joined to a larger high remission area that enveloped the West Coast, Southwest and central parts of Tasmania.

Total Number of Episodes of Cough

A low level of cough experience, one attack only, appeared to be an urban phenomenon; rural areas were significantly low (Figure 2.4.3). A history of two to five attacks was of above average incidence in the interior and Southwest parts of the

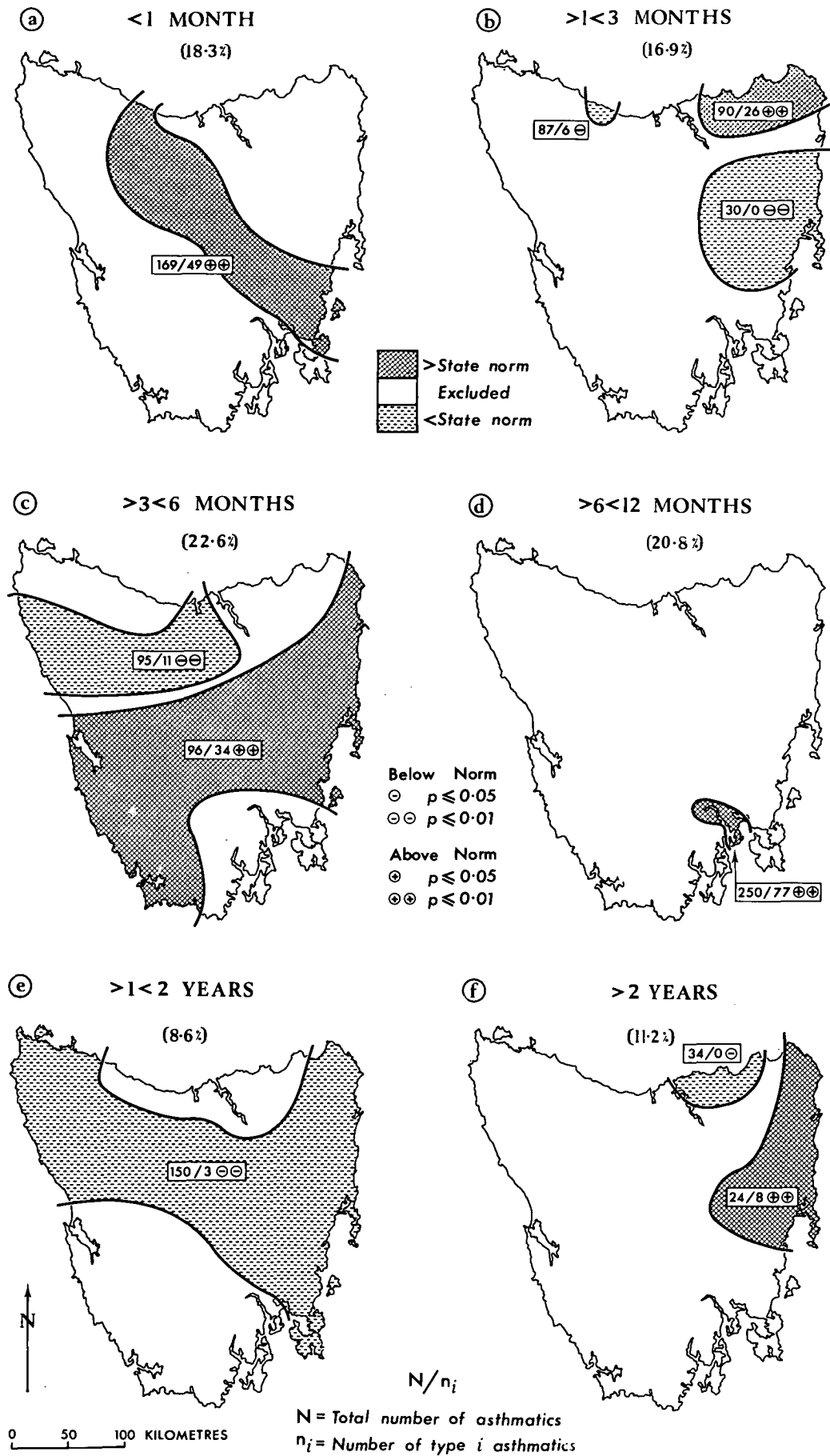


Figure 2.4.2 Time Since the Last Episode of Productive Cough

state, $p \leq 0.01$. This area was correspondingly low for those in the category of six to ten attacks, $p \leq 0.05$. Those who had experienced eleven to twenty attacks were low in a large rural area, and high in the Devonport locality, $p \leq 0.05$. Over twenty attacks were experienced more often in the Tamar region and were missing from the central and Northeast rural areas.

A history of two to five attacks showed a similarity to the pattern for an episode of cough between three and six months ago (Figure 2.4.2.c). Both maps portray a band of low incidence stretching from the Tamar to the West Coast and avoiding the Northwest. This can be explained by both the probability of late-onset coughers having had fewer attacks and the increased probability of late-onset coughers having had attacks more recently.

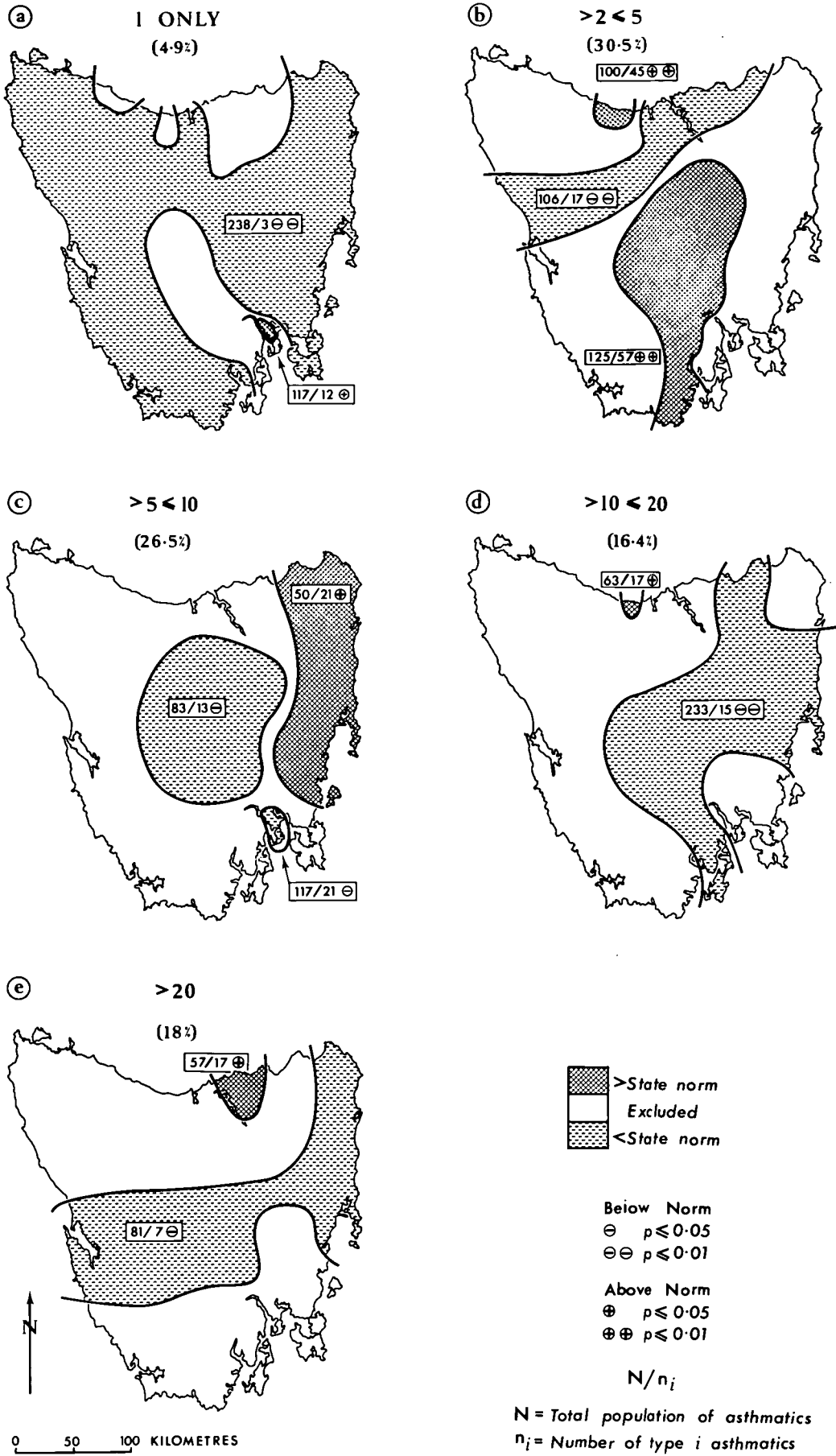


Figure 2.4.3 Total Number of Episodes of Productive Cough

2.5 Spirometry Variables

The great majority of cases were followed up with a clinical examination that included spirometry. From the spiromographs four measures of pulmonary efficiency were extracted; the vital capacity, the forced expiratory volume in half a second, the forced expiratory volume in one second and the maximum expiratory flow rate. Spirometry, especially vital capacity, is influenced by many factors: e.g. time of day, temperature and altitude. A child's height and sex are also important. Although it was difficult to adjust for the first three variables, each child's height and sex were available to standardise the data.

This was achieved by dividing an individual's spirometry values by their height² and taking the sexes separately. The spirometry to height ratios were then transformed to z-scores so that the four variables could be compared on the basis of their distributions about their respective means. The distribution of z-scores for each variable is illustrated in Figure 2.5.1. The histograms give the percentage distributions in half standard-deviation divisions. There are three histograms for each variable; one for each sex and one for the total population. Generally, the distributions were leptokurtic; for example, eighty-five per cent of the vital capacity/height ratios were within one standard deviation of the mean. The flattest distribution, that of the maximum expiratory flow rate/height ratio, had seventy-three per cent of the population within the span of one standard deviation.

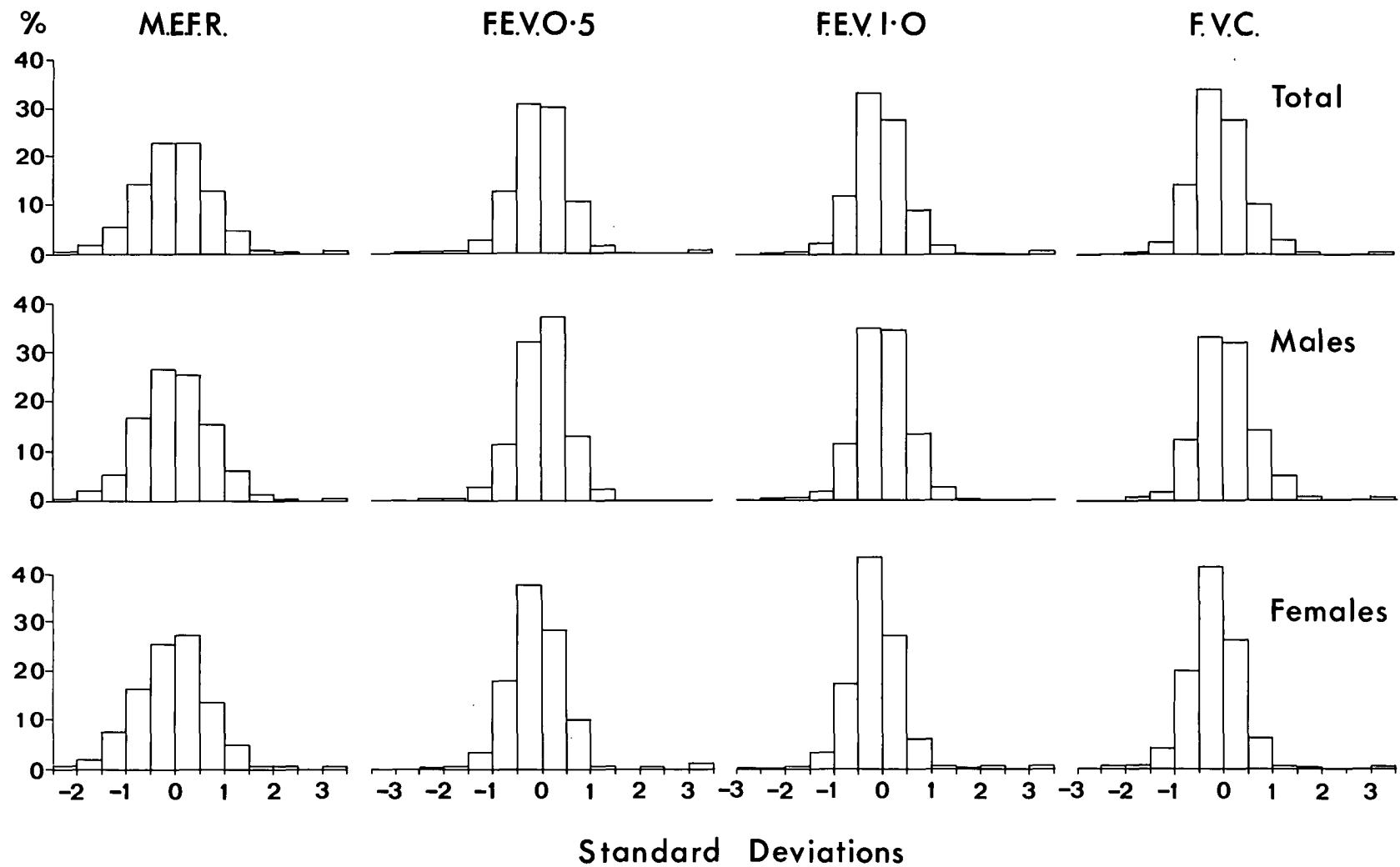


Figure 2.5.1 Frequency Distributions of Standardised Spirometry Scores

The distribution of z-scores for the sexes showed some dissimilarities. The significance of these differences due to sex were tested by using chi-square. The scores for the maximum expiratory flow rate to height² ratios were, however, quite alike. The absolute frequencies of male and female M.E.F.R./Height² scores gave an insignificant χ^2 of 4.18 with seven degrees of freedom. For the other three variables, the female scores were consistently skewed toward the lower tail of the distribution. This was most marked for the Vital Capacity/Height² scores which gave a χ^2 of 81.97 with seven degrees of freedom, $p \leq 0.001$. The forced expiratory volumes in half of a second and in one second gave a χ^2 of 29.1 and 42.8 respectively; both were significant at, $p \leq 0.001$.

Because of these sex differences and the unequal ratio of males to females in the primary schools, it was thought that the mapping of the combined sexes would introduce errors that could negate any statistically significant patterns. The sexes were, therefore, treated separately. The intention had been to look at the extremes of each distribution; to examine the spatial clustering of scores below or above one standard deviation from the mean. The leptokurtic nature of the distributions, however, made a half standard-deviation distance from the mean more useful as, in many cases, only a dozen individuals fell into any tail region greater than one standard deviation. For probability mapping, the cases were viewed as if they were described by a dichotomous binary variable; they could either belong to the category being examined or to the other (i.e. the rest of the distribution). The

TABLE 2.5.1
Spirometry Cross-Correlation Matrix

	F.E.V. $\frac{1}{2}$ sec.	F.E.V. 1 sec.	F.V.C.
F.E.V. 1 sec	0.90	-	-
F.V.C.	0.60	0.74	-
M.E.F.R.	0.84	0.87	0.65

figures are structured so that the tails are compared horizontally across the page and the sexes vertically down the page.

The spirometry variables examined here are unlike any of the variables considered previously. The questionnaire data examined earlier were subject to all the errors of memory recall and the individual variability in morbidity recognition. Spirometry gave an objective, interval-level, measurement of the consequences of respiratory morbidity. Furthermore, the data were collected close together in time and so represent an accurate picture of the state of pulmonary efficiency in the entire population for that given point in time. Because of the close interrelatedness of the four variables one would have expected any pulmonary insufficiency or pathology to be reflected in all of them. This was true; table 2.5.1 shows their correlation matrix. Some variables were more sensitive to respiratory complaint because of the intervening effect of an exogenous factor, for example, the F.E.V. $\frac{1}{2}$ sec. (because of the smaller volume of air to be expelled in total), was a more sensitive indicator of airways obstruction in the seven year olds than the F.E.V.1 sec. This was dependent upon the factor of time contained in this parameter.

After these considerations, the close parallels in patterns shown in Figures 2.5.2, 2.5.3, 2.5.4 and 2.5.5 were to be expected. Indeed, the variation between maps of the same sex and extreme could be due to the slight differences in absolute numbers mapped as a result of the shapes of the distributions.

A flattened distribution would have had more cases in its tails than a peaked one. When mapping small numbers these differences could easily have affected the spatial significance levels.

Maximum Expiratory Flow Rate (M.E.F.R.)

This variable stood out from the others in several ways. First there was no significant difference between the frequency distributions for the sexes. Second its frequency distribution was the flattest, hence more cases spread into the tails and more were mapped. Third, of all the spirometry variables, this measure was the one that was most independent of height, sex, or measurement parameters, it was a flow rate measured in litres/sec² from the steepest slope on the spiograph.

The sexes, however, demonstrated different spatial concentrations (Figure 2.5.2). Males having M.E.F.R. to Height² ratios less than -0.5 standard deviations from the mean were surplus in the Hobart area and the Tamar region. When the majority of the East Coast, the Southwest, the West Coast and the North Coasts were excluded, the rural interior parts of the state enjoyed a significant deficit of cases with this level of M.E.F.R., $p \leq 0.01$. This low-incidence, rural area was much reduced on the female map and did not include the Northeast. The Northwest, west, Southwest, and Southeast coasts, without the Hobart urban area, were correspondingly over-represented for females.

At the healthier end of the spectrum were those with M.E.F.R./Height² ratios greater than 0.5 standard deviations from the mean. Males enjoyed a surplus in a wide band stretching from the Northwest to the Southwest and including the central highlands,

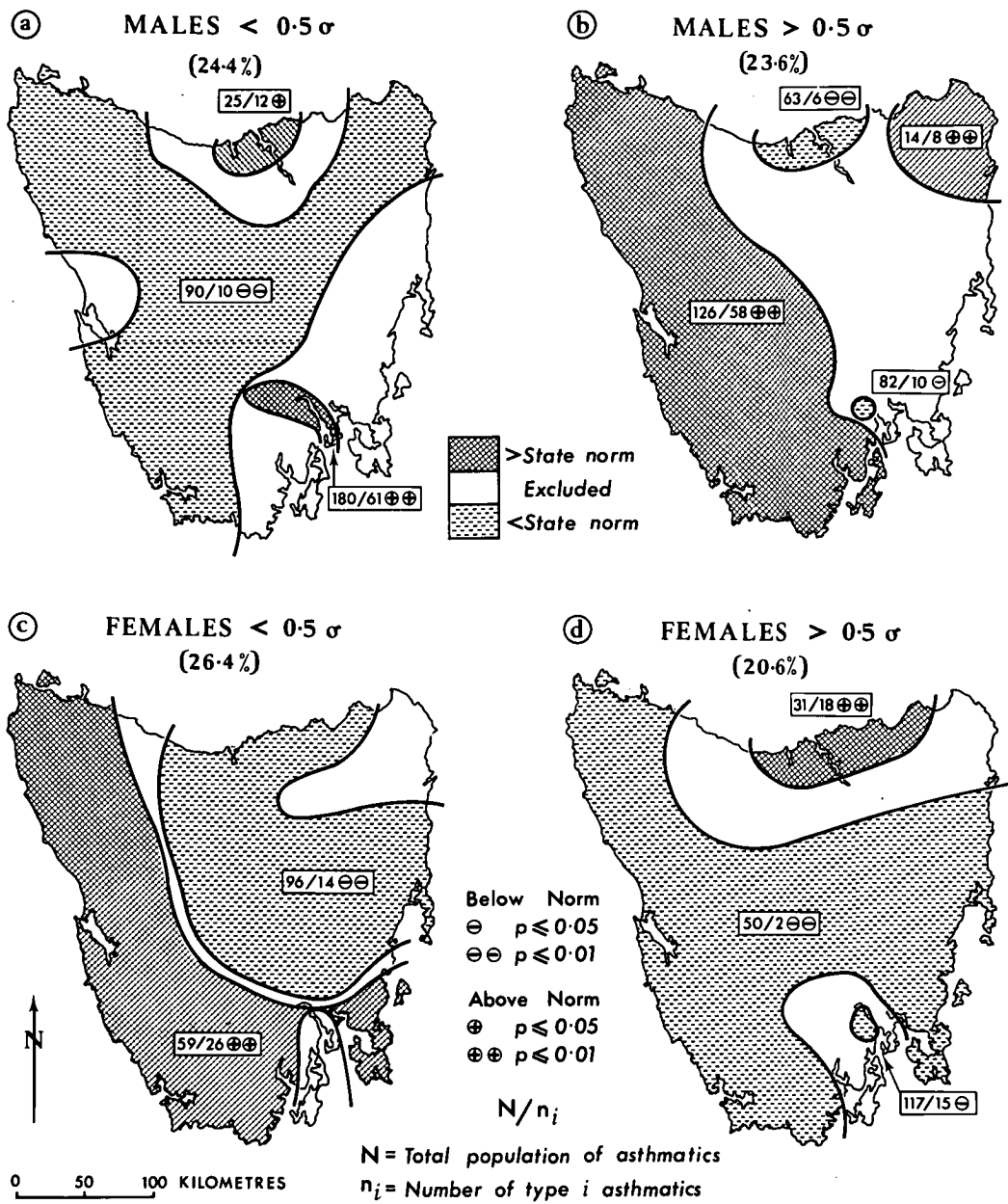


Figure 2.5.2 Maximum Expiratory Flow Rate/Height²

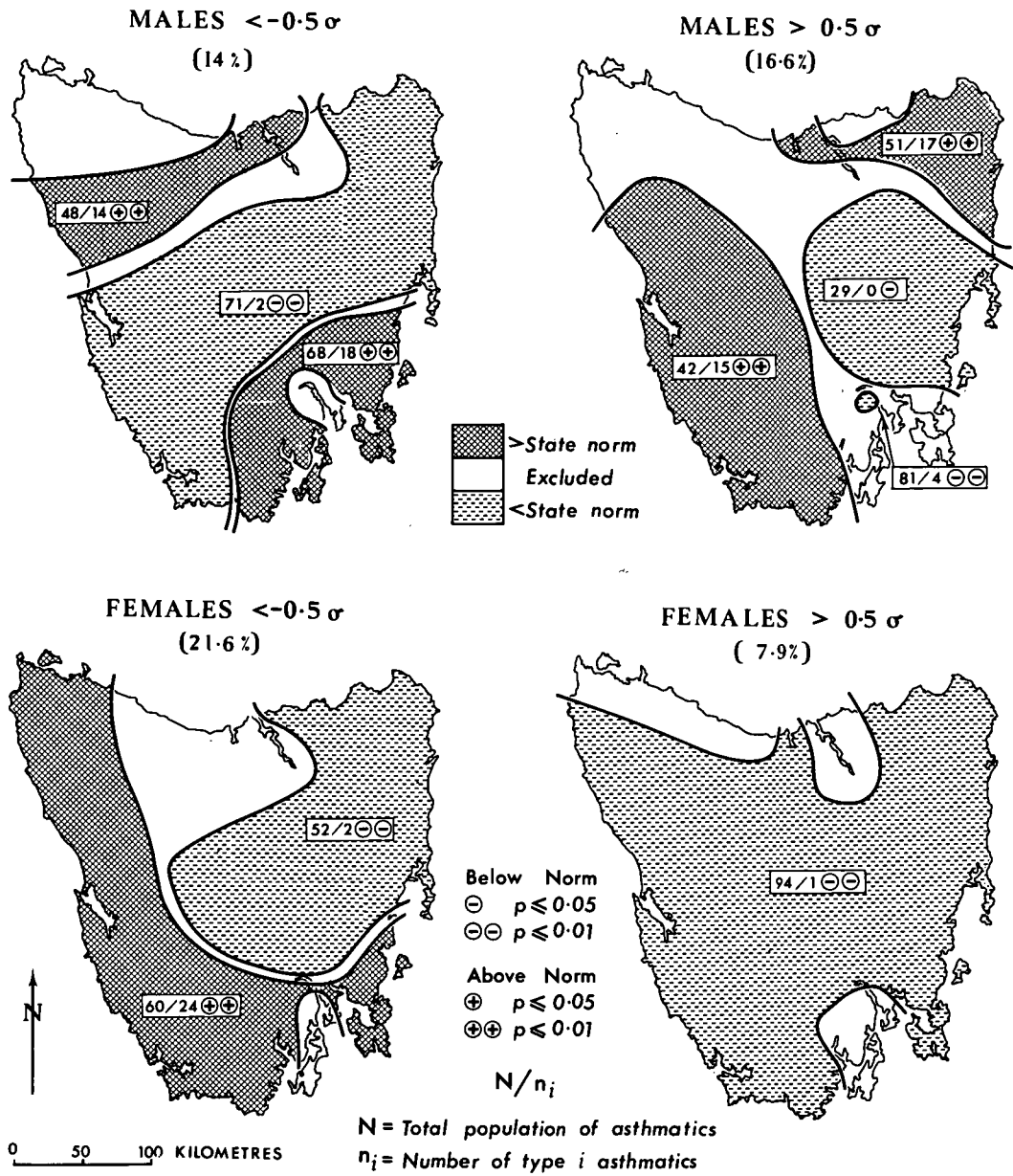


Figure 2.5.3 Vital Capacity/Height²

$p \leq 0.05$. The female pattern was almost the reverse that of the males. The Tamar region was high, $p \leq 0.01$, and the West Coast/Interior was low, $p \leq 0.01$. Hobart and Glenorchy together were low, $p \leq 0.05$.

Vital Capacity (V.C.) and Forced Expiratory Volumes (F.E.V.)

The spatial clustering of the high and low extreme values are illustrated in Figures 2.5.3, 2.5.4 and 2.5.5 for vital capacity, F.E.V. $\frac{1}{2}$ sec, and F.E.V.1 sec., respectively. The patterns bear a marked similarity and any differences are thought to be probably a function of the differing proportions of the population found in the different tail areas. The patterns for males and females are distinctly different, matching the differences in absolute frequency distributions.

The male pattern of low spirometry values showed a surplus of cases in an area that started on the Northern West Coast and included the hinterland to the Northwest Coast and ended on the central North Coast including Devonport and Georgetown. This area closely paralleled one found in the distribution of those with a history of productive cough three to six months previously (Figure 2.4.2.c). The other area high in prevalence of low values was the Hobart Metropolitan Area. This was surrounded by an area of average prevalence as was the northern high prevalence area. The mostly rural parts of the state were significantly low in low values. To an extent, the M.E.F.R. patterns were also in concordance with these. The main difference was that the northern area was limited to the Devonport/Georgetown area on the North Coast.

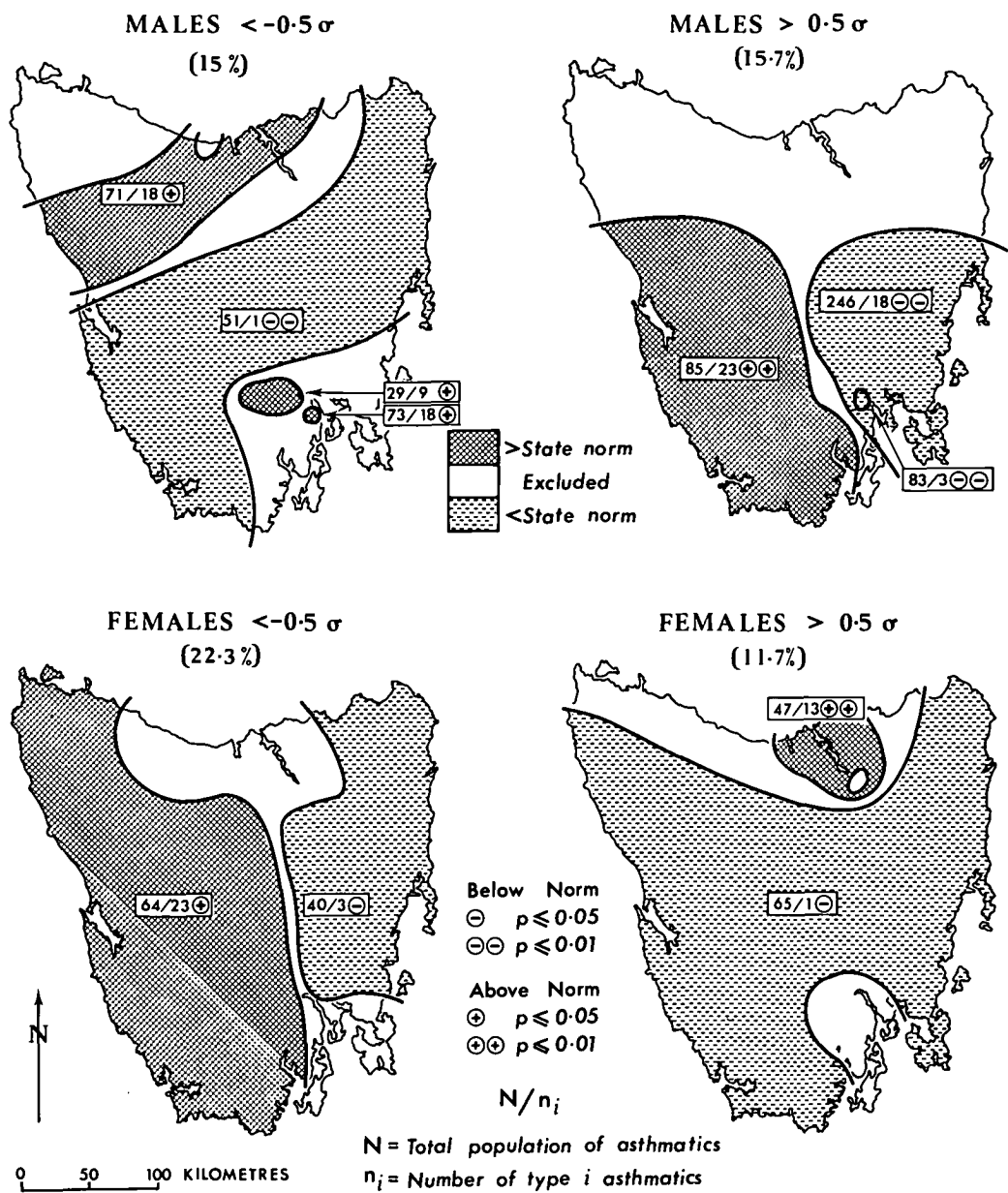


Figure 2.5.4 Forced Expiratory Volume in Half a Second/Height²

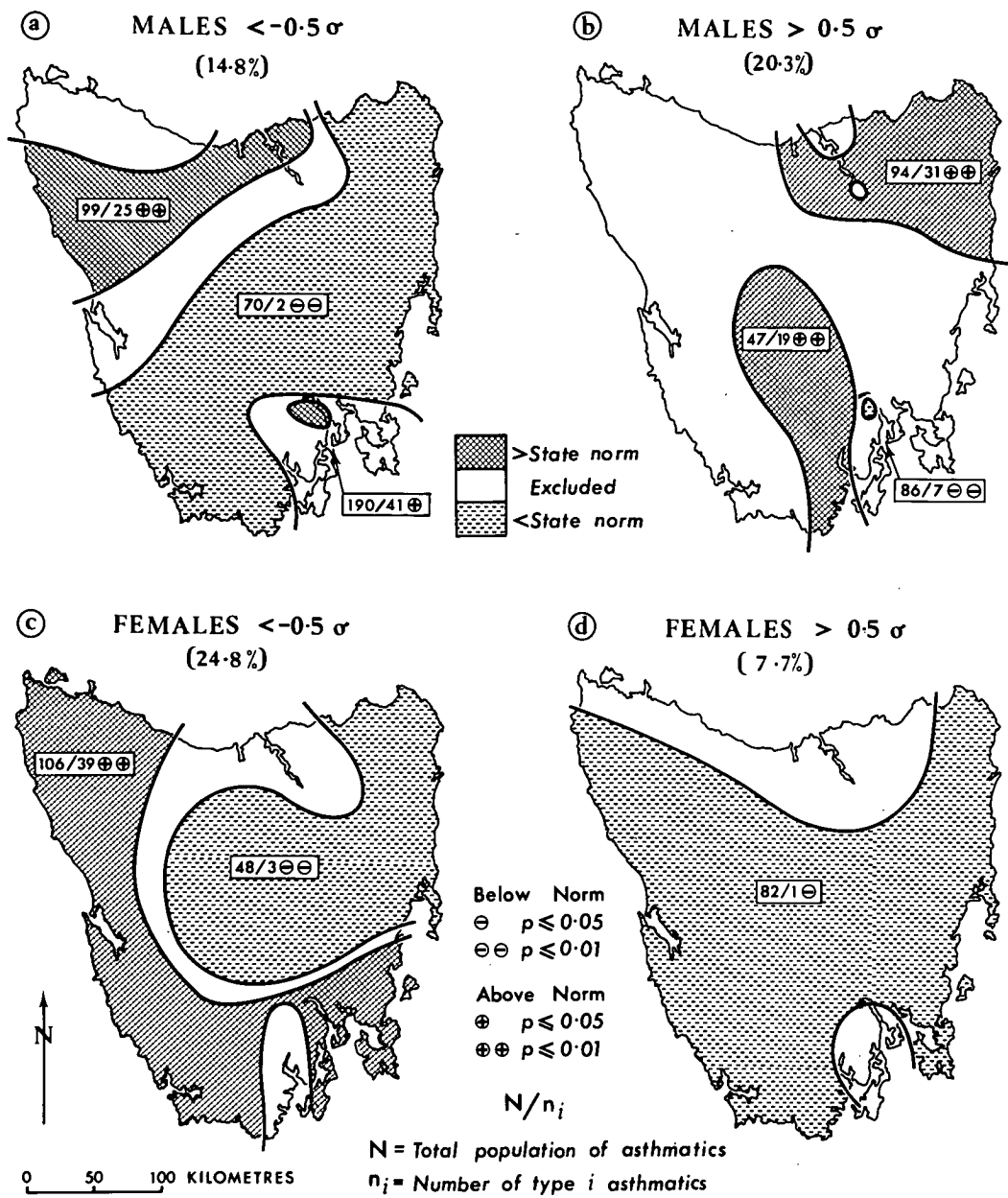


Figure 2.5.5 Forced Expiratory Volume in One Second/Height²

The patterns for low respiratory function in females were very similar; a high area of low values ranging from the extreme Northwest to the Southeast and an area devoid of low values that embraced the Midlands and the Northeast. In each area the urban centres of Hobart and Launceston were excluded because of their close-to-average levels. The strongest divergence from this pattern was that for females with low F.E.V._½ sec. values. Here, the western area of high prevalence encroached eastwards to include the central highlands, while the Midlands/Northeast low-prevalence area shrank back to the East Midlands, East Coast, and Northeast. Both areas were significant at, $p \leq 0.05$, whereas the other three maps attained levels of, $p \leq 0.01$.

Males having high spirometry values were prevalent in the Northeast, the central Midlands and most of the western half of the state. Exceptions to this pattern were vital capacity, where the West Coast area was limited to the Southwest and extended inland to include the Central Midlands, and M.E.F.R. where the West Coast area became most extensive and included the extreme Northwest. Low areas were found in suburbs of Hobart and in M.E.F.R. in the Tamar area. Also, for the F.E.V. variables, the East Midlands and East Coast formed a low prevalence area.

Healthy values in females contradicted, to an extent, those found for males. When the urban areas were removed, the predominantly rural parts of the state were found to be low, $p \leq 0.01$. For M.E.F.R. another low area was discovered in the Western Shore suburbs of Hobart, $p \leq 0.05$. Only two of the maps

gave significantly high areas. In both M.E.F.R. and F.E.V. $\frac{1}{2}$ sec., the Tamar region was high, $p \leq 0.01$, for healthy lung functions.

When all four variables are considered together their close correspondence of spatial clustering emphasises their meaningfulness and also underlines the differences between the sexes. This difference in spatial concentration suggests that aetiological and natural history dissimilarities exist between males and females. If a composite map of the four variables and the two tails were made on the basis of the healthy and unhealthy areas for males then the Northeast, central Midlands and Southwest would be healthy and the Hobart area, Southeast, Tamar and parts of the Northwest would be unhealthy. Unhealthy parts of the map for females would include most of the West Coast, the Southwest and Southeast and part of Hobart. Healthy areas would be the Tamar region, the Northeast and central Midlands. The female maps however, are not as consistent as those for males. This may be due to the smaller numbers involved.

If Figure 2.5.3 is taken as an example, unhealthy female values are significantly low in the Northeast/Central Midlands and healthy females are also significantly low in this area. The lack of extremely low values is not to be interpreted, therefore, as a sign of an abundance of healthy ones or vice versa as this may merely indicate a clustering about the mean. Only the positive surplus of low or high values should be taken as indicators of unhealthy or healthy areas. Recapitulating, considering only the positive surpluses, healthy areas for males would include the Northeast, West Coast, Southeast and the central

Midlands. Unhealthy areas would be Hobart, the Southeast, the Tamar and parts of the Northeast. Unhealthy areas for females would be the extreme Northwest, West, Southwest and the Southeast. Healthy areas would be the Tamar region and the central part of the North Coast.

2.6 Geographical Analyses of the Changes in 1961 Birth Cohort Between 1968 and 1974

Given that geographical variations could be identified in the prevalence of wheezy symptoms in 1968, it was hypothesised that the underlying factors influencing these differences might also have an effect upon the patterns of remission and new incidence of asthma in the cohort when it was re-surveyed in 1974. Such patterns would reinforce or suggest aetiological trends and causal hypotheses in different regions of the state. High positive or negative correlations between patterns could indicate either similar or different environmental risks and/or benefits.

Remission of Asthma Symptoms 1968-1974

In 1968, 857 boys and 535 girls were found to have histories of asthma/wheezy breathing. When re-surveyed in 1974 only 754 (88 per cent) boys and 475 (85 per cent) of the girls were still residing in Tasmania. The following is based, therefore upon that fraction of the cohort remaining in Tasmania. Of the 754 male asthmatics remaining in Tasmania 287 (38 per cent) enjoyed remission of symptoms during the period of six years; this compared to 39.4 per cent for females. Analysis of areal variations in proportion was accomplished by aggregating the cases by the primary school attended in 1968 and then grouping these schools into high and low clusters for the probability mapping technique described earlier. As the proportion of remissions was so high, the deviations from the norm were tested using the binomial distribution.

For males, an extensive area of high remissions stretched from the Northwest, through the central Midlands to the central East Coast. It avoided the Launceston area, the Northeast, the Hobart area and the South (Figure 2.6.1). The map for females was fairly similar. In this case the high remission area did not extend to the East Coast and a low prevalence area was discovered that included Burnie and Devonport and stretched westwards to the West Coast. Both sexes gave separate support to the observation that rural, and particularly higher-altitude, inland, areas enjoy a significantly greater proportion of remissions than urban and certain coastal areas in Tasmania. The Northwest is confusing. The "bad" effects of the industrial cities seem to be offset by some other factor influencing the distribution of male remissions as females did exhibit low remission rates in these areas.

The incidence of male asthmatics in this time period was low in the western half of the State and in the Northeast. Taken together, these areas gave an observed number of new cases that was significantly less than expected, $p \leq 0.01$. Although no significantly high areas were found for males, the central North-South axis of the state, sometimes described as the ecumene of Tasmania, gave above average numbers. Female new incidence was most noteworthy in the Northeast, where, the surplus of new cases was significant at $p \leq 0.01$. The Hobart Metropolitan Area was also high, $p \leq 0.05$. A rural area that included the central highlands and parts of the Northwest Coast was correspondingly low, $p \leq 0.01$.

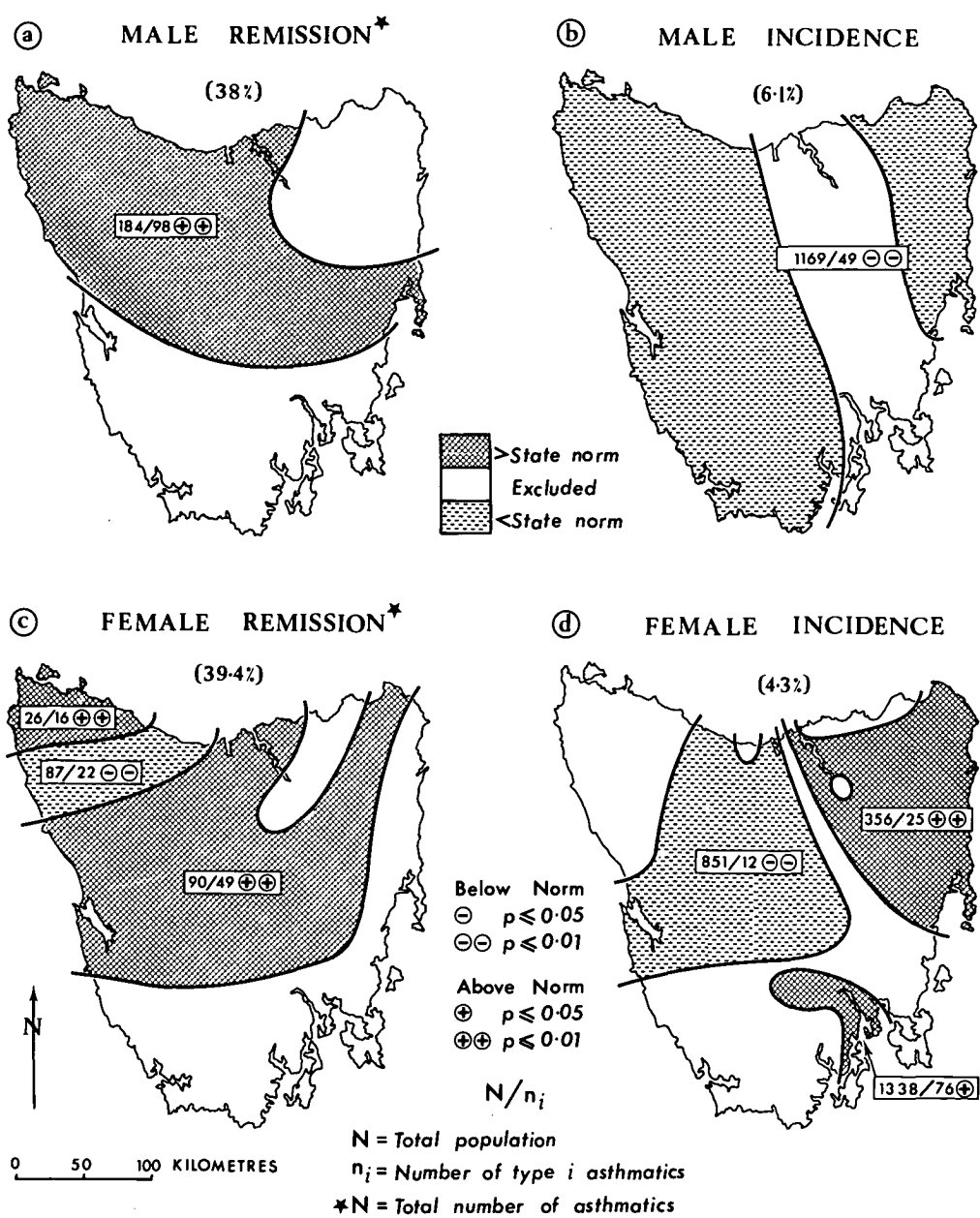


Figure 2.6.1 Remissions and Incidence 1968-1974

Summary

The patterns of incidence between 1968 and 1974 provided an interesting continuation to the maps of age of onset illustrated in Figure 2.3.1. The composite map (2.3.1.d) demonstrated a spatial progression of age of onset starting on the West Coast and moving North then East, then Southeast and eventually proceeding inland. When the four individual maps are examined in more detail it is noticed that the urban areas and the Northeast do not take part in this pattern. It took figure 2.6.1 to complete the picture. The maps of new incidence between 1968 and 1974 portrayed a significant surplus of females in the Northeast and in the Hobart Metropolitan Area and, although males were not significantly high, an above average band of incidence stretched from South to North from Hobart through Launceston to the North Coast. The "holes" in Figure 2.3.1.d were thus filled in. The male new incidence formed an obvious continuation of the high incidence area in 2.3.1.d and the female incidence redressed the balance in the cities and the Northeast sector.

A re-appraisal of Figure 2.2.1 emphasises this spatial progression. Map 2.2.1.a can be interpreted as the equivalent of the composite 2.3.1.e. Its importance is the significant deficit in total prevalence, in 1968, in the Northeast; and, in 1.2.1.b and 2.2.1.c. deficits in the Hobart area and in the Midlands. New incidence in the next six years was to remove these disparities.

One interpretation of the observed progression in incidence over time and space is to consider the manifestation of asthma

as similar to that of an epidemic in a susceptible population. If it is assumed that the proportion of asthma susceptibles would be fairly evenly distributed geographically, an "outbreak" of early onset cases as was noted on the West Coast would probably affect most of the population at risk in that area. The likelihood of a renewed incidence in the same area would, therefore, be remote. Future space-time clustering "epidemics" would be restricted to the areas where there were still large numbers of susceptibles.

This does not explain why the origin of the process was on the West Coast or its approximately clockwise progression about the state. To answer these questions one has to take into account the factor(s) responsible for transforming a latent disposition into an overt morbid condition. Differences between geographically discrete populations in regard to disease manifestation can be due to either differences in the subjects or in their environments or both. In other words, different areas will provide different triggers or levels of a given trigger factor. Differences in aetiology may be due to differences in degree as well as kind of insult. From another point of view, the latent asthmatics living in different environments may demonstrate variations in host-resistance. The threshold for a particular environmental stimulus morbidity response might be far different for a person living on the coast than for his counterpart living in the interior.

The concentration of late incidence asthma in urban locations can be seen to accommodate both of these concepts. Urban

places are usually markedly warmer than their surroundings due to the "heat island" effects and household heating is probably at its most modern and efficient in such areas. As cold is a known asthma stressor, any marginal increase in warming efficiency might mediate the harmful effects of cold and thus increase host-resistance to the disease. Conversely, urban areas possess the heaviest burden of atmospheric pollution. The sustained exposure to an "acceptable" level of suspended particulates or sulphur dioxide might prove sufficient to lower the average threshold for infections or other respiratory insults in an urban population. It can be seen that in any environment there are antagonistic elements that continually act upon the morbidity threshold and that, sooner or later, an asthma susceptible will have that threshold exceeded. However, in the case of asthma early onset seems to be related to a poor prognosis and an increased frequency of attacks related to an increase in bronchial hyperactivity. Preventive measures should thus be linked to delaying onset and thereafter reducing the number and mediating the effects of attacks. From this point of view, the late-onset areas would appear to be healthier environments.

2.7 Factor Analysis - One Approach Toward An Objective Taxonomy of Childhood Asthma

The results from the initial, relatively simple, mapping projects were sufficiently encouraging to continue the search for spatial differences and their causes. Brief perusal of the data, however, demonstrated immense variability between individuals. Indeed, in some cases, within-group variation could well have been higher than between-group variation. Several categories could be distinguished on the basis of only one or two parameters, for example, the total number of attacks experienced and the average length of time between attacks dealt with earlier. The 1968 survey, however, collected data on dozens of variables both from the health history questionnaire and a clinical examination. The arbitrary selection of a few characteristics for classification purposes seemed to be extremely wasteful of information.

In order to make better use of the total of available information in the data it was decided to take advantage of the generalising power of factor analysis.⁴ This technique can achieve considerable data parsimony while retaining a large amount of explanatory variance by creating a few linear constructs from the original variables. From the correlation matrix between the variables, factor analysis minimises the distance between co-varying variables on geometric planes of co-variance inherent in the data: the so-called factors. The factors are assumed to be the "real" underlying functions or processes that related variables imperfectly measure. Later it will be observed that a number of variables measuring the differing aspects of productive cough all load

highly on the same factor. This factor obviously describes the multivariate entity of respiratory infection and bronchitis. The first factor extracted accounts for the largest proportion of explained variance. The variables that load highly on each factor aid in its description. For this reason, the factor axes are often rotated to ensure maximal loadings of highly-related variables and minimal loadings of accidentally or non-related variables. The most commonly used rotation is the varimax solution used in this analysis. Eventually, each case can be given a score on each factor extracted. The factor scores can then replace the multiplicity of variable values. Furthermore, as the factors are extracted orthogonally they are independent of each other and can therefore be entered into regression analysis and other techniques that require this quality.

The computer programme used for this analysis was the sub-programme FACTOR included in S.P.S.S. version 7.⁵ In all, fifty-nine variables were taken from the survey master tape and after z-score standardisation were examined for suitability for analysis. Initial analyses soon identified unsatisfactory, redundant or poorly-measured variables. In the final factor solution, twelve variables satisfied the criteria of normality, communality and theoretical meaningfulness. These were as follow in Table 2.7.1.

Preliminary to the factor analysis was the correlation matrix. Table 2.7.1 shows the correlations between the variables greater than ± 0.1 . Correlations less than this are given only as plus or minus signs signifying the direction of relationship. The twelve variables entered were reduced to three factors that accounted

TABLE 2.7.1
CORRELATION MATRIX

	1	2	3	4	5	6	7	8	9	10	11
1. Last Episode of Wheezing											
2. Frequency of Wheezing Episodes	.58										
3. Total Episodes of Wheezing	-.41	-.31									
4. Last Episode of Cough	.46	.28	-.26								
5. Duration of Cough	+	+	-	.68							
6. Onset of Cough	-	+	-.16	.57	.75						
7. Total Episodes of Cough	-	-	.24	.31	.81	.59					
8. Height	+	+	-	+	+	+	-				
9. Forced Expiratory Vol.½	+	+	-	+	-	+	-	.48			
10. Forced Expiratory Vol.1	+	+	-	+	-	+	-	.48	.90		
11. Vital Capacity	+	+	-	+	-	+	-	.62	.69	.74	
12. Maximum Expiratory Flow Rate	+	+	-	+	-	+	-	.42	.84	.87	.65

Coefficients <0.1 are given as + or -

for the major part of the explanatory variance in the data.

Table 2.7.3 gives the eigenvalues and proportion of variance attributed to each factor and the loadings of each variable, on each factor.

As in the correlation matrix, only loadings greater than ± 0.1 are shown. Smaller loadings are again indicated by plus and minus signs.

An examination of the factor loadings gives clues as to what the underlying dimensions represent. The loadings can be interpreted much as regression weights: they indicate the contribution that each variable makes to the factor and the sign gives the direction of influence the variable imposes on the dimension.

Factor 1-Spirometry Factor

This factor loads highly and positively upon the four spirometry variables and height. High scores on this function will be achieved by cases having high spirometry scores; low score by those possessing low spirometry. From figure 2.7.1 it is seen that the low tail of the distribution of scores on this factor is both extended and thickened. Below -2 standard deviations lie about nine per cent of the cases; an unusually large proportion for an otherwise fairly normal distribution. These are the severe cases with either permanent crippling of the lungs or very recent episodes of respiratory distress. As would be expected, barely one per cent have scores above two standard deviations greater than the mean.

Table 2.7.2

Varimax Rotated Factor Matrix

Variables	Factor 1	Factor 2	Factor 3
Height	0.56	+	+
Forced Expiratory Volume $\frac{1}{2}$ sec.	0.92	+	+
Forced Expiratory Volume 1 sec.	0.96	-	+
Vital Capacity	0.79	+	+
Maximum Expiratory Flow Rate	0.87	-	+
Duration of Cough	+	0.97	+
Onset of Cough	-	0.75	+
Total Episodes of Cough	+	0.83	-0.19
Last Episode of Cough	-	0.71	0.47
Last Episode of Wheezing	+	+	0.86
Frequency of Wheezing	+	+	0.64
Total Episodes of Wheezing	-	-	-0.52
Eigenvalue	3.49	2.79	1.58
Percentage of Variance	44.4	35.5	20.1

Coefficients <0.1 are given as + or -

Factor 2 - Cough Factor

Here, the loadings are both high and positive on the four variables entered in the analysis that described the experience of productive cough. This factor, thus, provides a scale upon which the degree of productive cough can be measured. It will be noticed from the figure that the frequency distribution is bimodal. The peak in the 2.5 to 3 standard deviations class that accounts for about eleven per cent of the population, represents those cases without any history of productive cough. The remaining, negatively skewed, distribution represents the spectrum of coughers. A high score on this factor signifies the absence of cough; a low score *vice versa*.

Factor 3 - Remission Factor

Both cough and wheeze variables are implicated in the structure of this factor. The highest loading is on the time since the last episode of wheezing. The higher the value for this variable the longer the time that had elapsed since the last attack. A positive loading on this variable, therefore, represents a tendency to remission of symptoms. This is true also for positive loadings on the time since the last episode of cough and the average frequency of wheezing. The negative loadings on the total number of episodes of both cough and wheeze point further to a mild, easily-remitting condition hence the label 'remission factor'; rather than "wheeze factor". Very low scores on this factor indicate chronic, refractory asthma that implies a poor prognosis.

Each case was given a standardised score on each of the factors. The distribution of these z-scores is given in histogram form in figure 2.7.1. As has already been described; the spirometry factor scores were overly supplied in the low tail; the cough factor scores were bimodal thus isolating the non-cough population and the remission factor scores formed a relatively flat, symmetrical distribution with equal numbers outside of the one standard deviation limits. Only cases in these extreme tails were chosen for mapping on the rationale that the extremes would be more revealing than the norms found within a standard deviation of the mean. The percentages of each sex found in each tail of each factor are given in table 2.7.4. These represent the state norms against which regional variations in proportion were tested during probability mapping. Community prevalences less than five per cent were tested against the Poisson distribution; larger proportions were modelled on the binomial. The results are displayed in Figures 2.7.2 to 2.7.4.

A caveat must be noted in regard to the interpretation of the relationship between high and low incidence areas when the category mapped represents one tail of a normal-like distribution. As was noticed in the interpretation of spirometry values for females in a previous part of this section; a significant surplus of members belonging to the extreme tail under examination is a tangible, positive, estimate of the spatial clustering of that variable. Negative clusters, extremely low prevalences, are interesting but are strictly only relevant to the spatial distribution of members of that tail. The absence of members

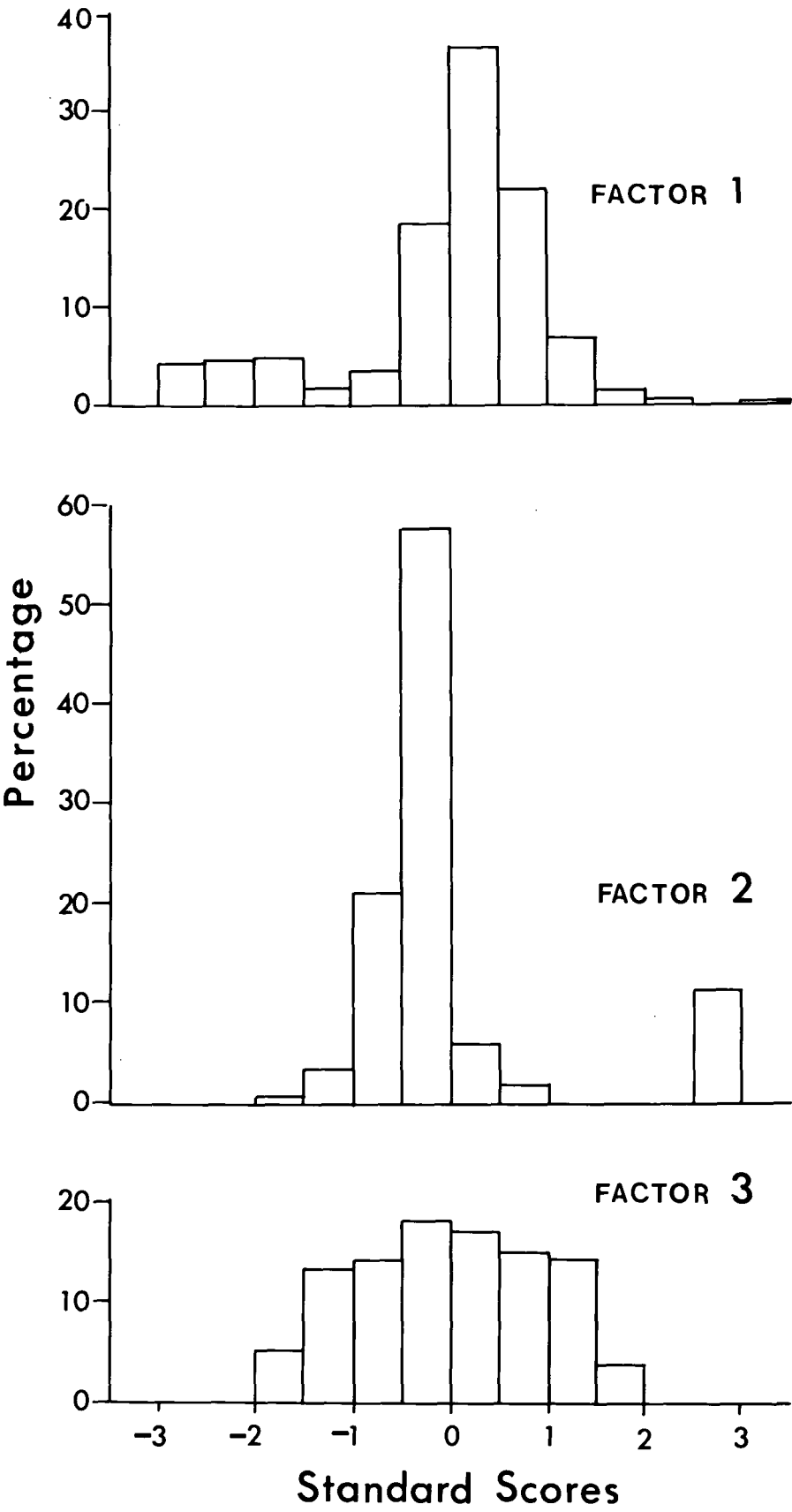


Figure 2.7.1 Percentage Distribution of Standard Factor Scores

Table 2.7.3

The Percentage Distribution of Factor Scores by Sex

Factor	Scores above or below a Standard Deviation from the Mean					
	Males (840)		Females (547)		Total (1387)	
	$<-\sigma$	$>\sigma$	$<-\sigma$	$>\sigma$	$<-\sigma$	$>\sigma$
Spirometry	14.4	9.9	12.4	4.6	13.6	7.8
Cough	3.5	11.0	3.1	10.6	3.3	10.9
Remission	17.9	15.5	17.4	20.5	17.7	17.4

of one tail does not necessarily imply an abundance of members of the opposite tail: it may very well be due to an excess of average cases. This situation is peculiar to the interpretation of the patterns arising from a consideration of the two extremes of a symmetrical distribution. It does not apply, of course, to a single binary situation of presence or absence. In that case the absence of one type obviously implies the presence of the other.

The extremes of the spirometry factor are mapped for the two sexes separately in figure 2.7.2. It was expected that spatial patterns would show some congruence with those for the individual spirometry values mapped earlier. This appeared to be more or less the case. Male cases with low spirometry scores gave a closely similar spatial clustering pattern to that for low M.E.F.R. scores (Figure 2.5.2.a). Males with high spirometry scores matched their spatial distribution to that of males with high scores for F.E.V. $\frac{1}{2}$ sec. (Figure 2.5.4.b). Females with low spirometry scores had a pattern that matched quite well with that for female F.E.V. $\frac{1}{2}$ sec., (Figure 2.5.4.c), but differed in the extent of the two opposed areas. High female spirometry values had a spatial clustering surprisingly close to that for female vital capacity (Figure 2.5.3.d) and F.E.V.1 sec., (Figure 2.5.5.d), differing only in that the Hobart/Channel area gained a moderate significance, $p \leq 0.05$. The spirometry factor was thereby, seen to serve well as a reliable composite of the four original variables and thus to serve also as their surrogate.

The cough factor proved to be more difficult to compare with individual variable spatial patterns as maps had only been

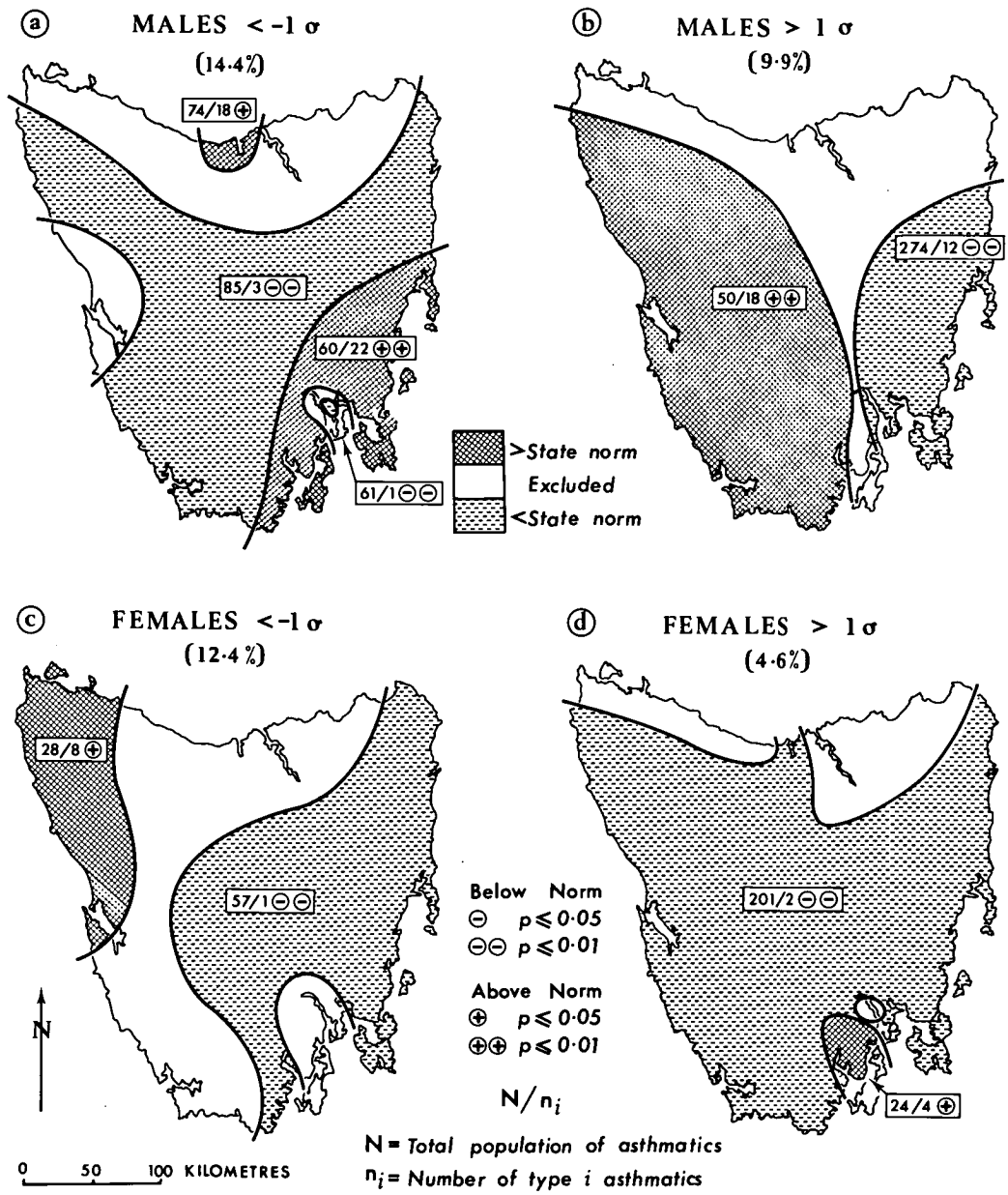


Figure 2.7.2 Spirometry Factor Scores

produced for the total number of episodes of productive cough experienced, (Figure 2.4.3), and the time since the last episode of cough, (Figure 2.4.2.), neither of which treated the sexes separately. The greatest correspondence was found between the spatial pattern of males with scores more than one standard deviation above the mean (cases without history of cough) and the map of wheezers without cough, (Figure 2.2.1.d). This was not surprising as the populations were identical. The low scores for males (severe cough) were conspicuous, spatially, by their absence from the Southwest central Midlands and Northeast. The same was true for females in this category, though their low area was more extensive than that for males. In the higher categories of the total number of episodes of productive cough (Figure 2.4.3.c., d. and 3), low prevalence areas are shown that cover parts of the cough factor's deficit areas. As these categories are linked to the more severe cases of cough their parallel spatial patterns supported the meaningfulness of factor cough as a substitute for the original variables.

Factor 3 was labelled the 'remission factor'. If this label was to be meaningful, scores on this dimension should have differentiated between chronic cases with refractory symptoms and those which had enjoyed good health for some time. The key variables in this factor are the time since the last episodes of wheezing and cough. Other indicators of remission were the variables that described the average frequency of episodes of wheezing and cough. Each of these variables had a category for no attacks in the last two years, that is, remission. Once again, comparison

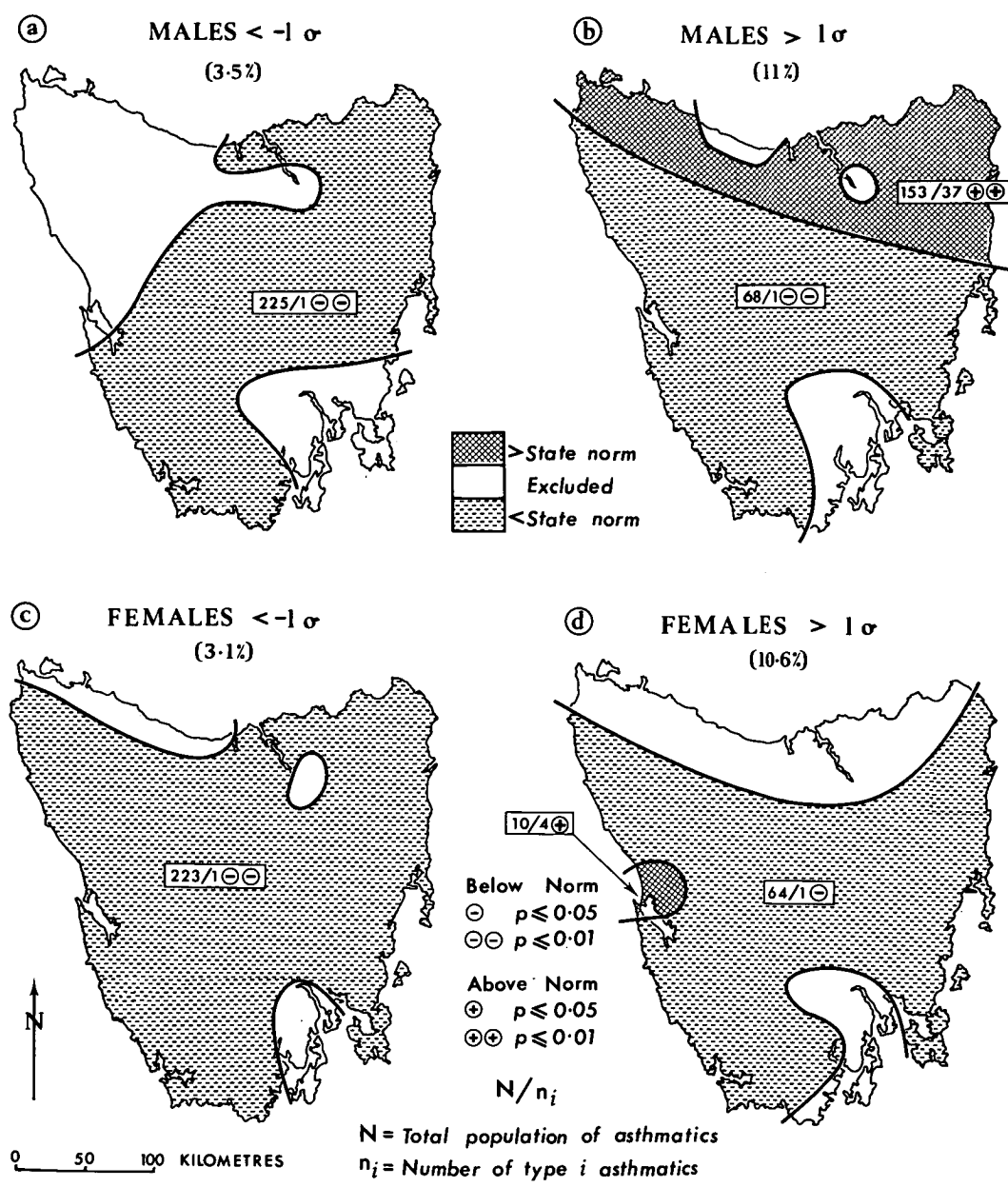


Figure 2.7.3 Cough Factor Scores

with single variables was made difficult by the lack of separate mapping for each sex. However, figure 2.3.6.d shows high levels of remission in the Northeast that agree with the distribution of male remissions in figure 2.7.4.b and this agreement is also found with the last episode of productive cough being more than two years ago (Figure 2.4.2.e). The refractory male cases distribution is matched by figure 2.4.2.a; males having had a productive cough within the last month. The high remission area for females is also common to the pattern in figure 2.3.2.d (last episode of wheezing more than two years ago). It was concluded, on this basis, that the remission factor truly represented the dimension of remissivity.

This analysis achieved two major objectives. First an unwieldy number of variables was reduced to a small number of orthogonal, independent, linear constructs that could serve either as substitutes for the original variables in further analysis or as objective yardsticks with which to measure and/or compare new cases, samples or populations. The factors, as generalisations of health status, can be used in much the same way as clinical parameters to assess the morbidity level of an asthmatic. Second, the spatial variations shown in individual variable distributions of extreme factor scores. Spatial clustering detected in the proportion of remissions or severe cough may serve to generate hypotheses with respect to the influence of environment on disease patterns. For, if different measures of the same morbidity parameter repeatedly show similar spatial environmental clustering, the suggestion of environmental interaction cannot be ignored.

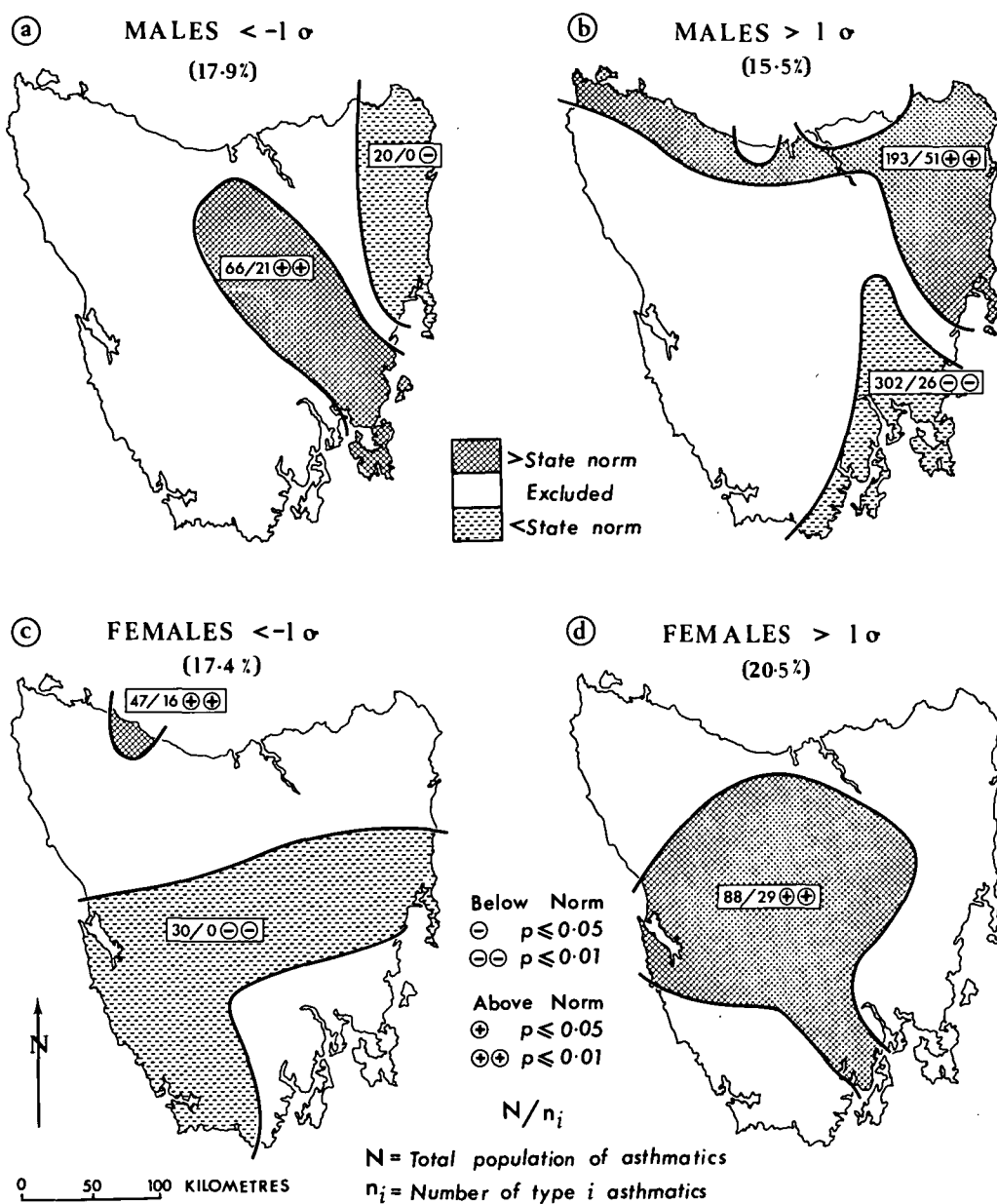


Figure 2.7.4. Remission Factor Scores

2.8 Geographic Patterns Arising from a Combined Agglomerative/ Hierarchic-fusion Cluster Analysis of Asthma Symptoms

Introduction

The previous chapter in this section described the use of factor analysis in the derivation of statistical parameters that could be used to differentiate the various subsets within the asthmatic population. This approach to classification viewed the problem from the interrelationships between the symptom variables. The correlation matrix was used to fashion additive linear constructs from the original variates. The factors accounted for the majority of the variance in the data matrix and could thus be used as surrogates for the original variables as case descriptors. Furthermore, considerable data parsimony was achieved in that twelve variables were reduced to three factors with minimal losses in explanatory power.

The classification described here takes a different approach; instead of looking at similarities between symptoms it looks at the net similarities between individual cases based on their total symptoms. The object of cluster analysis is to cluster cases into groups such that the degree of association between members of the same group is maximised and the degree of association between members of difference groups is minimised. Twenty interval or ratio level variables were chosen for the analysis (see Table 2.8.1). After standardisation by Z-score transformation to reduce the unwarranted effects of variables possessing large variances, the data was subjected to principal components analysis. The first ten components represented over ninety per cent of the

TABLE 2.8.1
Variables in the Analysis

Variable Description	Mean	Standard Deviation
Time elapsed since last episode of wheezing	3.50	1.76
Frequency of occurrence of wheezing	5.72	1.95
Average length of episode of wheezing	2.08	0.90
Age of onset of wheezing	2.58	1.66
Total number of episodes of wheezing	2.77	1.33
Time elapsed since last episode of cough	3.81	1.81
Frequency of occurrence of cough	5.07	2.35
Average length of episode of cough	2.21	1.13
Age of onset of cough	2.32	1.74
Total number of episodes of cough	2.69	1.55
Height in cms.	118.49	13.79
Forced expiratory volume $\frac{1}{2}$ sec.	1151.21	247.91
Forced expiratory volume 1 sec.	1449.04	336.49
Vital capacity	1621.58	343.06
Maximum expiratory flow rate	1917.19	537.67
Composite score history and presence of allergies	0.47	0.63
Composite score history and presence of wheezing	1.02	0.19
Composite score history and presence of cough	1.65	0.54
Composite score history and presence of upper respiratory infections	1.03	0.89
Composite score history and presence of eczema	0.88	0.48

variance in the data. The standard scores on each of these components were used as the input to the clustering procedures.

The Analysis

The CLUSTAN 3 package of subroutines was used to effect the cluster analysis. The maximum number of cases that could be processed using this software was 1000. Because of this the sexes were clustered separately. Two analyses were performed; one for boys (690) and one for girls (458). The restriction in the number of cases proved to be beneficial as it necessitated making a comparison of the results and, hence, an assessment of the reproducibility and robustness of the resultant classification. Given more than one sample, the lack of discernible differences between the different solutions gave a strong indication of a robust, local-optimum classification.

The first clustering procedure used iterative relocation. This method initially allocates individuals to k clusters sequentially. Then, during each relocation scan each case is considered in turn and the similarity between it and each of the clusters is calculated using the error sum of squares method. If the similarity of a case with a cluster q is higher than with its parent cluster p then the case is relocated to cluster q . The population is repeatedly scanned until no cases are relocated during one complete scan. A local optimum solution for k clusters will have been reached at this point. Next the similarities between all pairs of clusters are calculated and the pair having the highest similarity are fused together forming $k-1$ clusters. The relocation phase is then repeated until a local optimum

solution is found for k-1 clusters and then fusion to k-2 clusters takes place etc.

The strategy adopted for this analysis was to allocate the cases sequentially to twenty groups. Using five relocation phases, the local optimum solutions were found for each cluster number from k-1 to k-5. The terminal solution gave fifteen stable clusters for input to the next phase. This was to fuse the k-5 clusters hierarchically down to k-19. No relocation took place; the cases were restricted to their parent clusters. In each fusion cycle the two most similar groups were joined. The similarity measure for this process was the same error sum of squares statistic used for the relocation procedure. This is the sum of the distances from each case to the centre of the cluster to which that case belongs. Thus it measures the extent of scatter about the cluster centres. It is most suitable for finding tight clusters with the property that each cluster centre represents the constituent cases at a high level of similarity with respect to all of the underlying variables.

Error Sum of Squares for cluster (p)

$$E_p = \frac{1}{M_{ipj}} \sum \sum (X_{ij} - U_{jp})^2$$

Where -

M is the number of variables

X_{ij} is the i'th observation for variable j

U_{jp} is the mean of variable j for the cases comprising cluster p

The male and female cluster analyses used 690 and 458 cases respectively. This difference in numbers resulted in different error sum of squares for the two samples, however, the sequence and structure of the fusion hierarchy was unaffected. Figure 2.8.1 illustrates the dendrograms for each sex with the error sum of squares information statistic measured on the vertical ordinate. The dendrograms clearly demonstrate the similarities between the two analyses.

The Clusters

Many of the fifteen initial clusters were shown to be very close together in the lower levels of the dendrograms. As fusions increased the aggregated clusters became more distinct. When the fusion coefficients were graphed for each clustering, discontinuities or jumps in the information statistic were detected. In this way one could distinguish meaningful, interpretable clusters from statistically significant, but poorly understood, groupings. A graph for each sex was given in figure 2.8.2. From each of the analyses five distinct clusters were obtained. The means for each of the twenty variables are given for each cluster for each sex in tables 2.8.2.a and b. Although some minor differences were observed none of them were statistically significant. The five clusters from each analysis possessed the same structure; an indication of robust classification. An examination of tables allowed one to interpret and describe the clusters that had been obtained. The means located the group centres and were thus fair descriptors of the clusters.

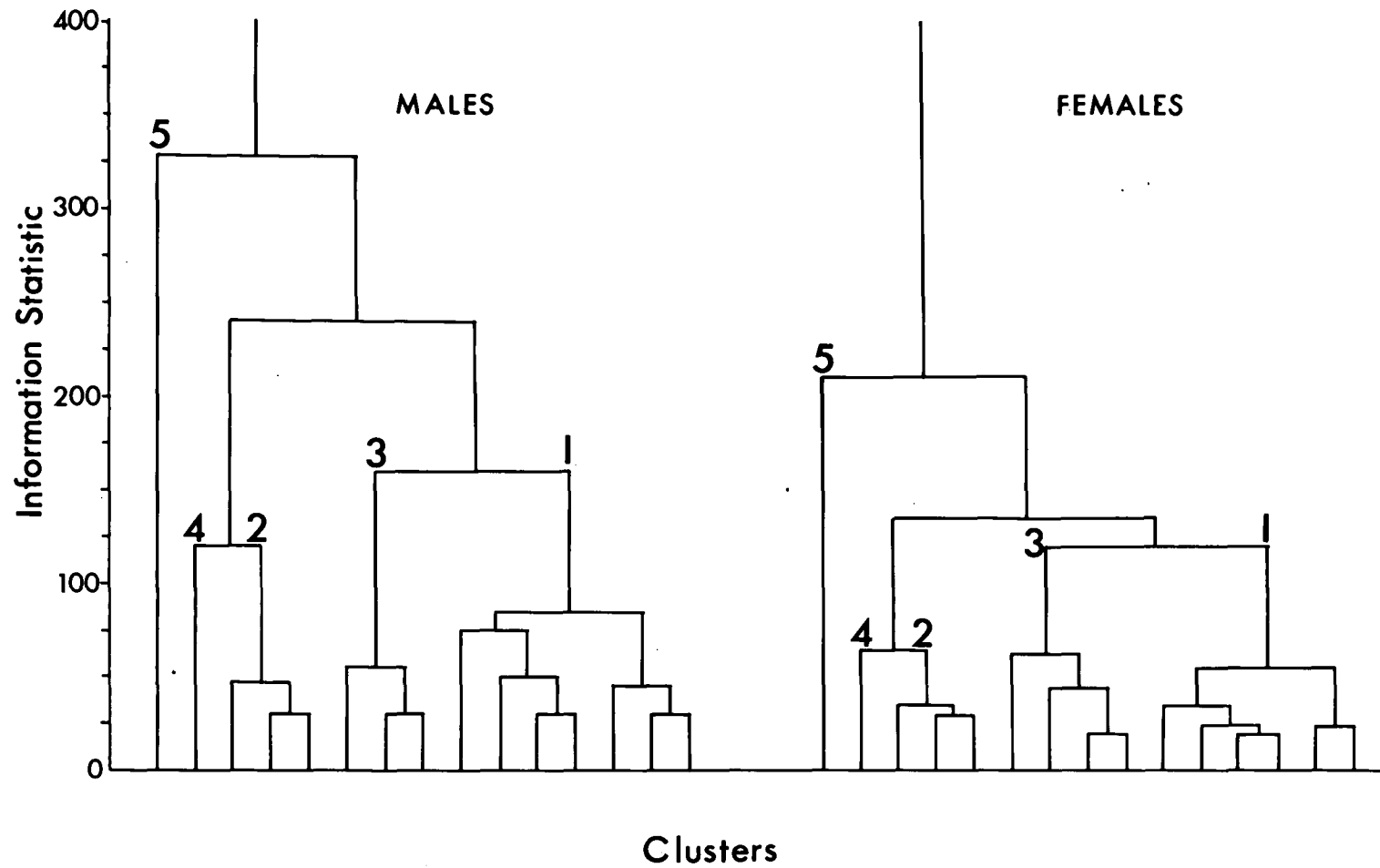


Figure 2.8.1 Dendrograms of the Clusters Obtained for Each Sex

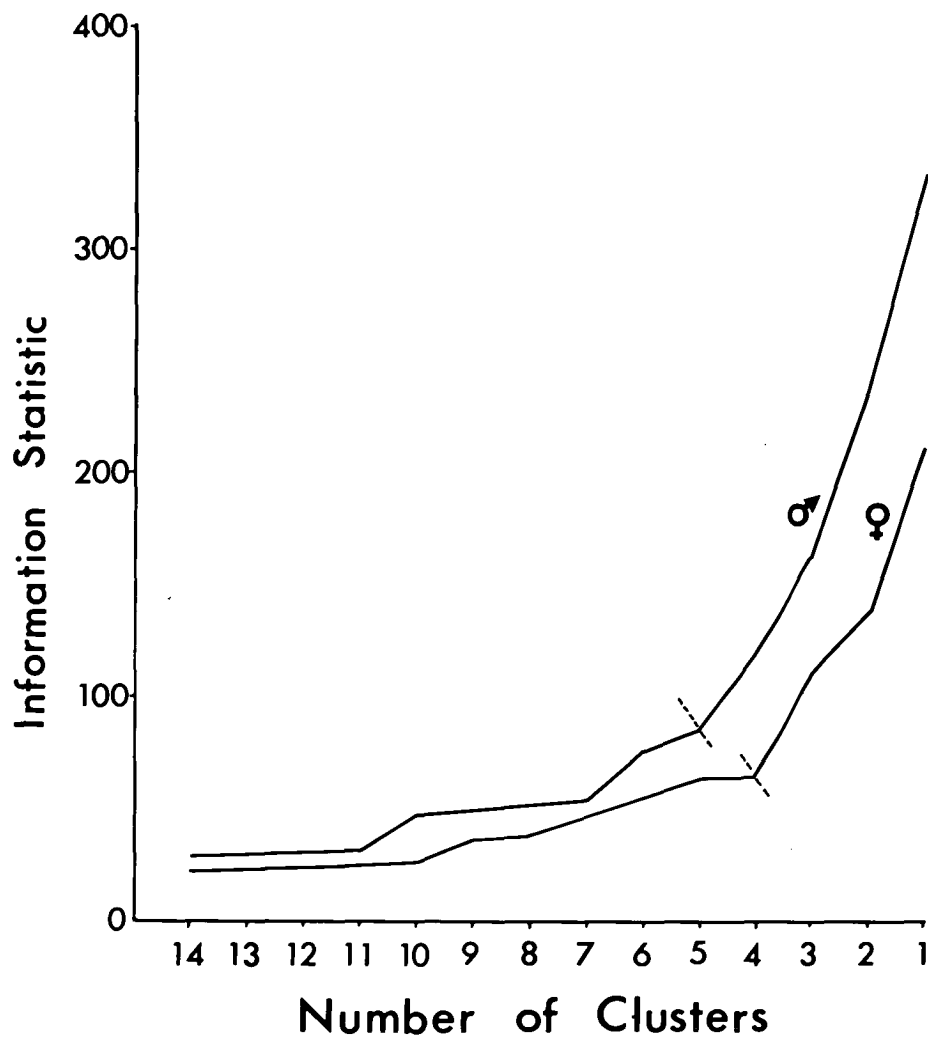


Figure 2.8.2 Cluster Fusions

TABLE 2.82a&b

Variable Means for Each Cluster by Sex

(a) Males						(b) Females					
Cluster	1	2	3	4	5	Cluster	1	2	3	4	5
Variables						Variables					
1	2.9	2.1	5.1	3.1	3.9	1	3.4	1.6	4.5	2.6	4.5
2	5.6	4.4	7.3	5.4	5.8	2	5.9	3.9	6.6	4.9	6.4
3	2.1	2.4	1.9	2.3	1.8	3	2.3	2.4	1.9	2.1	1.9
4	3.8	1.9	1.6	1.9	3.0	4	3.2	2.7	2.1	2.8	3.2
5	2.7	4.1	2.4	3.6	2.5	5	2.7	4.2	2.5	3.8	2.2
6	2.9	2.1	4.3	2.5	-	6	3.2	1.8	3.9	2.3	-
7	5.7	4.6	6.9	5.0	-	7	5.7	4.4	6.4	4.4	-
8	2.5	2.6	2.3	2.5	-	8	2.6	2.8	2.3	2.3	-
9	3.7	2.0	1.8	1.9	-	9	3.1	2.9	2.3	2.5	-
10	2.8	4.2	2.6	3.3	-	10	2.9	4.2	2.7	3.5	-
11	121.9	117.8	120.4	120.4	121.2	11	122.4	116.3	118.2	118.3	120.5
12	1259.3	1034.3	1194.4	971.7	1175.2	12	1292.9	999.9	1014.0	951.8	1139.1
13	1615.3	1302.6	1506.5	1292.1	1468.3	13	1580.3	1254.9	1268.5	1200.2	1385.8
14	1826.8	1504.5	1194.4	1598.0	1681.6	14	1719.6	1409.4	1432.9	1419.7	1544.4
15	2086.1	1663.7	2007.3	1476.8	1920.1	15	2227.9	1608.4	1690.7	1499.1	1917.9
16	0.6	0.7	0.2	0.8	0.3	16	0.3	0.7	0.6	0.5	0.3
17	1.0	1.0	1.0	2.0	1.0	17	1.0	1.0	0.9	2.0	1.0
18	1.8	1.8	1.8	1.1	0.9	18	1.8	1.7	1.9	1.3	0.7
19	1.1	1.2	0.8	1.0	0.5	19	1.3	0.9	1.1	1.0	0.8
20	0.4	0.9	0.4	1.1	0.3	20	0.2	0.6	0.6	0.8	0.5

1. Last wheeze; 2. Wheezing frequency; 3. Wheeze duration; 4. Age of wheezing onset; 5. Total wheezes;
 6. Last cough; 7. Cough frequency; 8. Cough duration; 9. Age of cough onset; 10. Total coughs;
 11. Height; 12. F.E.V.₁ sec.; 13. F.E.V.₁ sec.; 14. Vital capacity; 15. M.E.F.R.; 16. Allergy score;
 17. Wheeze score; 18. Cough score; 19. U.R.I. score; 20. Eczema score.

Cluster 1 Late Onset - Bronchial Asthma

Had the highest age of onset for both wheezing and productive cough. This late age of onset was reflected in the highest values for height and spirometry. This cluster also had the highest mean values for history of productive cough and upper respiratory and throat infections.

Cluster 2 Early Onset - Moderate Bronchial Asthma

Had the highest average number and the longest duration of attack of both wheezing and productive cough. Persons belonging to this cluster possessed the lowest average height. Girls, in addition, had the lowest mean for vital capacity. Low mean values for F.E.V._{1/2} sec., and the low value for the time elapsed since the last attack completed the picture.

Cluster 3 Remissions

This cluster had the highest average time since the last episodes of both wheezing and productive cough; and the highest value for periodicity of attack (the higher the value, the longer the period). The total number of attacks was commensurately low. The highest mean for history of productive cough and the lowest for age of onset pointed to an early-onset wheezy cough with annual (probably winter) episodes. The large value for time-since-last-episode may be due, in part, to the time of year when the data was collected and the annual periodicity of this form of wheezing.

Cluster 4 Early Onset - Severe Chronic Asthma

This group was similar to, but more extreme than, cluster 2. Highest mean values for history of allergies, eczema and wheezing

occurred in this group. The lowest mean values for F.E.V. $\frac{1}{2}$ sec., F.E.V.1 sec. and M.E.F.R. indicated irreversible pulmonary effects.

Cluster 5 Non-Bronchitic Asthma

Characterised by zero values for variables measuring productive cough, this group, in addition, possessed the lowest mean values for duration of attack and history of cough, upper respiratory infections, eczema and allergies.

The distribution of cases into clusters by sex is given in table 2.8.3. The proportion of cases falling into each category was not significantly different between the sexes (χ^2 of 6.74 with four degrees of freedom). One in ten wheezers showed no signs of productive cough. Two in ten were chronic bronchial asthmatics. Three in ten were enjoying remission of their symptoms. Of the remaining four tenths nearly all had a mild form of late-onset asthma (36.5 per cent). Only one in thirty three (three per cent) fell into the most severely affected group.

Spatial Analysis

The spatial distribution of each cluster can be examined from two points of view. First, the number of asthmatics in each cluster can be calculated as a proportion of the total population of asthmatics and normals residing in a given area. This can be made more specific by taking the sexes separately. This proportion then represents the cumulative prevalence of a particular type of asthmatic generated in the cohort population

TABLE 2.8.3

Proportion of Asthmatics in Each Cluster by Sex

	Males		Females		Total	
	No.	%	No.	%	No.	%
Cluster 1	256	37.1	163	35.6	419	36.5
Cluster 2	152	22.0	74	16.2	226	19.7
Cluster 3	185	26.8	165	36.0	350	30.5
Cluster 4	21	3.0	12	2.6	33	2.9
Cluster 5	76	11.0	44	9.6	120	10.5

in a given time period (seven years). At the state level, taken as the norm for this analysis, all these prevalences fall beneath five per cent of the total population of either sex. The distribution of sample proportions can then be expected to follow a Poisson distribution and regional deviations from the state norm can be tested for statistical significance. Table 2.8.4 gives the respective prevalence rates by sex and total populations.

Second, the total population to be examined can be restricted to the asthmatic population. Ignoring absolute prevalence, each type of asthma will be expected to occupy a fixed proportion of the asthmatic population close to the state norm for that type. As can be seen from table 2.8.3, the proportion attributed to each cluster at a state level varies from three to thirty-six per cent. The distribution of sample proportions is best estimated, in this case, by the binomial distribution with the exception of cluster 4.

The locational information available for each case was the primary school of attendance in 1968. The number of cases in each cluster was cross-tabulated by school and sex, as was the total population. Given this information, three objectives remained to be gained.

- (a) The detection of significant variation in the relative proportion of each cluster in geographic samples
- (b) The detection of significant variation in the absolute prevalence of each cluster in geographic samples of the total population

Table 2.8.4

Community Prevalence Rates for Each Cluster by Sex

	Males		Females	
	No.	%	No.	%
Cluster 1	4404	5.81	4126	3.95
Cluster 2		3.45		1.79
Cluster 3		4.2		3.99
Cluster 4		0.47		0.29
Cluster 5		1.72		1.06

- (c) The detection of significant variation in the areal distribution of clusters from each sex.

These objectives were realised by probability mapping techniques similar to those used previously. The results are summarised in figures 2.8.3 to 2.8.5.

Cluster 1

Late-onset asthma deviated from its normal prevalence in several areas. Two high prevalence regions were significant at $p \leq 0.01$; they were an area around Devonport in the North and another peripheral to Hobart in the South. The West and far Northwest were deficient in late developers at $p \leq 0.01$. Although other parts of the state had values above and below the mean, none were significantly so.

Cluster 2

The proportion of early-onset, moderate, bronchial asthmatics showed little geographic variation. The only area that was at all deviant from the state norm was a large rural section that included the Midlands and the Northeast. Here the prevalence was low, $p \leq 0.05$.

Cluster 3

Cases enjoying remission of their symptoms were under-represented in the North Midlands, Tamar Estuary and Northeast, $p \leq 0.01$. The Midlands and East Coast, however, had a slight surplus of thesefortunates, $p \leq 0.05$.

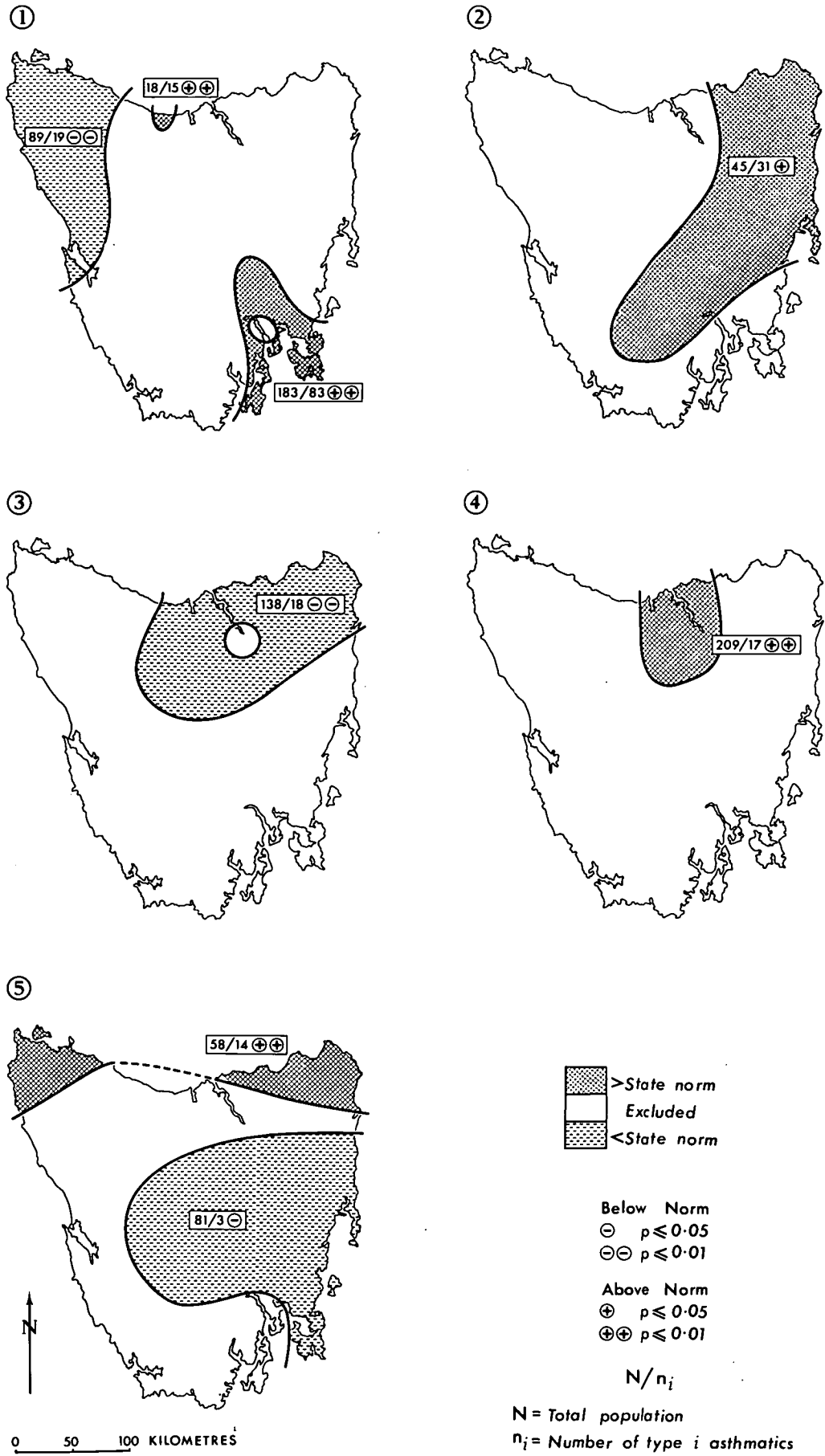


Figure 2.8.3 Spatial Variation in the Relative Proportion of Each Cluster

Cluster 4

The most severely affected asthmatics were most prevalent in a wide area around the Tamar Estuary in the North of the state. The proportion of cases in this area was over twice the norm; this was significant at $p \leq 0.01$. All cases were concentrated in urban settlements in this region and in the rest of the state. The southern half of the state contained only a quarter of this group; all others resided North of latitude 42 S. The city of Glenorchy was unusual in that no cases of this type lived there. It is suspected that children with this severity of illness were sufficiently ill to merit intra-urban migration to sites that parents perceived to possess a better standard of environmental health.

Cluster 5

This cluster contained about ten per cent of all the asthma sufferers. Children belonging to this group differed from all other groups in that they did not share the common characteristic of a history of productive cough. A latitudinal effect was thought to be observed in the distribution of these cases but was disturbed by an urban factor. In the predominantly-rural, northern parts of the state this type of asthmatic was twice as prevalent as would have been expected, $p \leq 0.01$. In the rural southern part of the state its prevalence was about a third of the norm, $p \leq 0.05$. Urban and other areas differed insignificantly from the norm.

The other two objectives were dealt with simultaneously by separating both the total and the various morbid populations

by sex and then testing for sex and cluster-specific, absolute-prevalence, deviations from the state averages. This was done by using the Poisson distribution as all the state norms were less than five per cent. Given the state's male and female populations, N_m and N_f respectively, and the number of cases belonging to each cluster by sex $x_1 \dots x_5$ the norms were calculated by dividing the male or female members of each cluster by the relevant populations:

$$\frac{x_1 \text{ males}}{N_m}, \quad \frac{x_2 \text{ males}}{N_m} \quad \text{Etc.}$$

These are given in table 2.8.4. From any geographic sample two numbers can be obtained for each sex and cluster combination; N and x_i . The x_i becomes the observed number of cases in the cluster. The expected number x_j is calculated by multiplying N by the respective norm calculated above. The observed number is looked up in tables and if the expected number lies within the limits for that value the deviation is insignificant. If the expected number was greater than the upper limit for the observed number it was significantly low and if the expected number was less than the lower limit for the observed number the observed number was significantly high. The process of aggregating schools has been described previously.

Summary

The significant patterns arising from this analysis are portrayed in the maps contained in figures 2.8.4 and 2.8.5. Asthmatics of either sex belonging to clusters 2 or 4 (the most severely affected groups) possessed fairly similar spatial concentrations. Areas peripheral to Launceston, the Tamar Estuary and the Northeast were prominent with respect to these types. Sex differences were discernible in the distribution of the other clusters. Cluster 1, the late onset cases, had above average numbers of females in the northeast corner of the state. Males, on the other hand, were surplus in the centre of the island and in the centre of the North coast. Remissions, cluster 3, were lower than expected for males in the Southeast of the state and, although higher than average, the male remissions on the Northwest Coast were insignificant. Female remissions were significantly low on the Northwest Coast and in the North generally. Most remissions in females were in the Hobart Metropolitan Area. Females demonstrated no significant patterns at all for cluster 5, non-bronchitic asthmatics. Males, however, were concentrated in the northernmost corners of the island.

The repeated patterns of incidence in the North of the state and in the central, "continental" areas strongly suggest climatic influences upon the distribution of certain types of asthmatic. This seems to be particularly true of cluster 5 members: these asthmatics who lack any underlying bronchitis are more prevalent in the lower latitudes. Patterns due to these

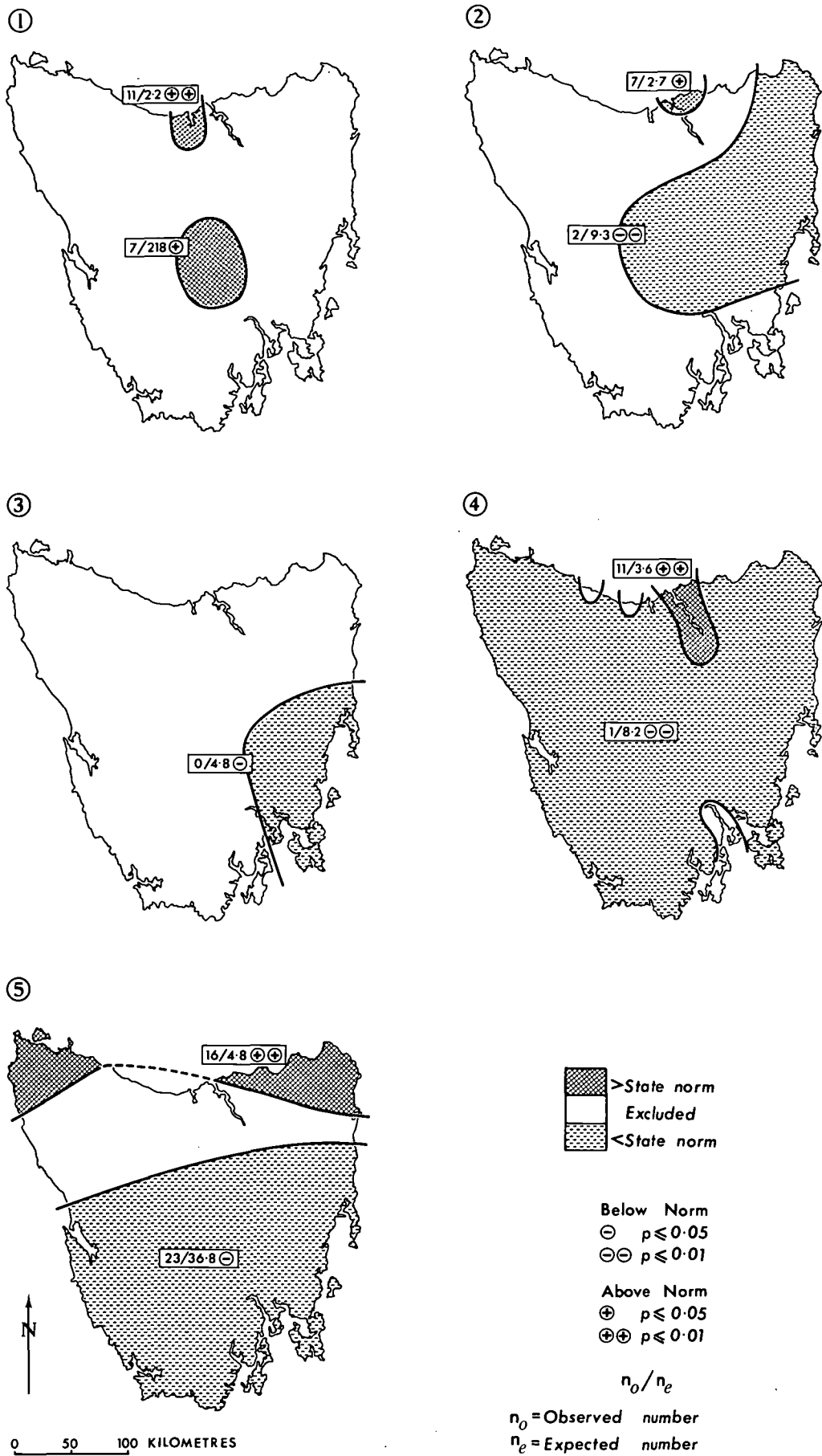


Figure 2.8.4 The Spatial Distribution of Males from Each Cluster

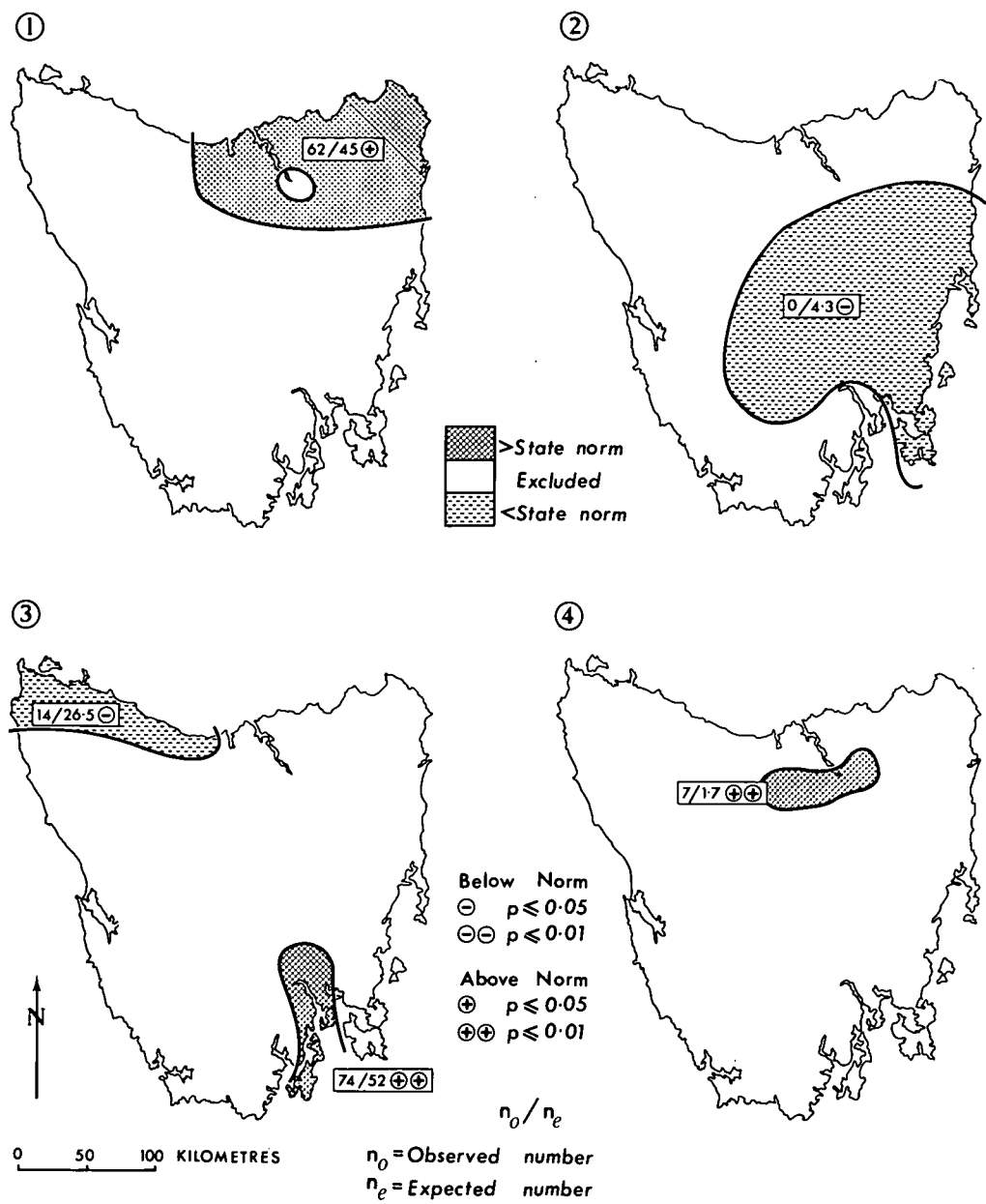


Figure 2.8.5 The Spatial Distribution of Females from Each Cluster.

physical factors seem to suffer distortions due to urban effect(s). This may be related to pollution and other urban stresses or may be just an artifact of migration.

2.9 The Uses of Discriminant Analysis to Define Geographic Types of Asthmatic Natural History

The two preceding analyses first looked at the interrelationships between symptom groups and second looked at interrelationships between groups of individuals. Both successfully described complementary aspects of asthma morbidity; the first by constructing dimensions upon which to measure asthmatics and the second by clustering asthmatics into statistically distinct clusters. This third multivariate approach to the cohort data set is an attempt to distinguish between and to classify individuals based upon the group experiences of individuals living in certain parts of the state.

It was hypothesised that sufferers from an environmentally sensitive condition like asthma who spent their maximum period of residence in dissimilar geographic areas would demonstrate differences in morbidity. Furthermore, a comparison of areal differences in morbidity would help identify the recognizable environmental elements. The research therefore centred around three objectives:

- (a) the selection of discrete, homogeneous geographic areas
- (b) the comparison of group characteristics of asthmatic individuals in those areas, and
- (c) the identification of environmental variables that differed between the areas.

Multiple discriminant analysis⁶ was chosen to identify geographic types within the wheezy breathing/asthma syndrome. This technique is designed to separate two or more groups using

a set of variables upon which the groups should theoretically differ. Mathematically, the intent is to force the groups to be statistically distinct by linearly combining the variables using different weighting coefficients for each group. Discriminant analysis actually serves two purposes; analysis and classification. The analysis phase involves the sequential selection of discriminator variables and the testing of the contribution of each successive variable for statistical significance. In this way the researcher includes only those variables that are useful discriminators discarding any that are redundant or useless. Similarly the number of discriminant functions can be limited to those that produce a significant increase in discriminatory power. In a two group example, one function is derived. When there are more than two groups more than one function is usually necessary. The maximum number of functions is one less than the number of variables.

Discriminant functions are very similar to factors extracted from a factor analysis: they are geometric dimensions upon which the cases are located. The weighting coefficients are also analogous to those in multiple regression or factor analysis: they identify the variables which contribute most to the differentiation along their respective discriminant function. The variable loadings can thus be used to indicate the nature of the functions which can then be examined for theoretical meaningfulness.

Once a group of variables has been found that can discriminate between individuals of known group affiliation a set of classification coefficients can be derived for each group. For

any case, these coefficients calculate a score for the probability of membership to each group and the case is assigned to the group for which it has the highest probability value. The classification process enables one to test the efficiency of the discriminant functions. When cases from known groups are allocated by the classification functions, the percentage of misclassifications demonstrates the degree of success or failure. If the allocation is successful individuals of unknown group membership can be confidently classified using the same functions; the level of confidence depending upon the proportion of misclassifications deemed acceptable.

Group Definition

Before the discriminant analyses could be executed the groups to be discriminated had to be defined. It was decided to start with a large number of small, relatively homogeneous physical environments and in the course of successive analyses to discard any areas with unacceptable proportions of misclassifications among their residents. In this way only a few strong types were expected to remain at the conclusion.

Twelve areas were selected for the initial analysis (Figure 2.9.1). This exercise drew a great deal upon detailed local knowledge from many sources. Places were included on the basis of popular reputation, general practitioners' opinions or asthmatics' anecdotes. Others were chosen for their unique climatic and physiographic characteristics and some for their urban-industrial pollution. Much of the information available was not supported by hard data. The best data obtainable were

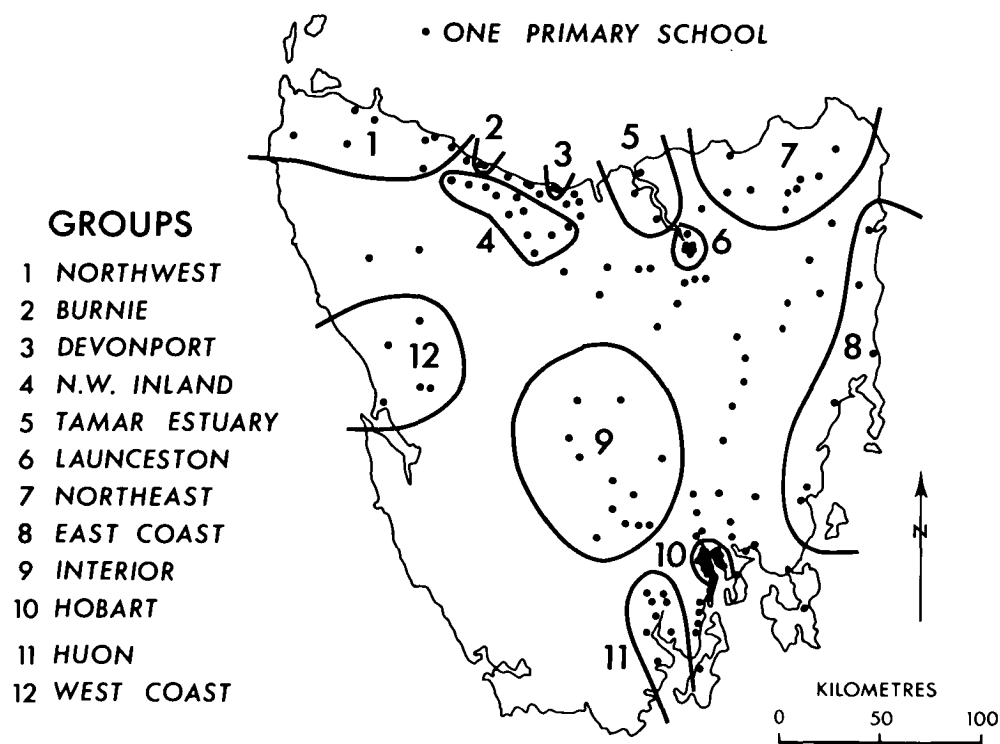


Figure 2.9.1 Areas Selected for Discriminant Analysis
(Source Areas)

physical-climatic averages. Some of these are given in table 2.9.1 to illustrate the climatic variation within the state. Each area is described by meteorological data collected from a representative, usually the only, station within its boundaries.

Although each of the areas shown on the map possesses a unique mix of climate, topography, industry and social structure, it was suspected that in some of the areas the within group variance would be greater than the between group variance and that some groups, therefore, would not serve as strong geographic types. Some areas, for example, will attract more in-migration than others and their population averages will be based on variable environmental histories and hence regress toward the state norm. Alternatively, many locations will have grossly-similar physical environments and will again tend toward a norm.

Analysis

The twelve groups entered in the initial analysis were a first approximation. The intention was to conduct more than one analysis and to discard any area that was unsatisfactorily discriminated. After four separate analyses, at the end of each, the two worse-classified groups were deleted, the final analysis was conducted upon four groups. The groups entered in this analysis were: the Northwest, the Interior, the Huon and Hobart. These four residential environments contained the extremes of the ranges of altitude, latitude, urbanisation and continentality within the state.

The discriminant analysis chosen was a step-wise model. That is, the best discriminating variable was chosen first, and

Table 2.9.1

Physical Characteristics of the Representative Meteorological Station in Each Area

	Altitude (metres)	Rainfall (cms)	Raindays	Max.Temp. (°C)	Min.Temp. (°C)	Range† (°C)
Northwest	4.6	92.9	201	16.1	9.2	21.0
Burnie	7.3	100.5	162	16.8	8.3	22.4
Devonport	12.2	92.4	138	16.7	8.0	23.3
Inland N.W.	269.7	120.9	143	15.6	5.7	24.6
Tamar	15.2	86.5	148	17.0	8.2	24.6
Launceston	106.7	71.4	131	17.4	7.2	27.2
Northeast	199.9	98.4	143	16.5	7.1	26.1
East Coast	30.5	68.4	99	17.7	9.4	19.6
Interior	349.9	55.1	140	16.5	3.7	32.5
Hobart	55.2	62.2	162	16.8	8.3	24.6
Huon	39.9	87.8	149	16.9	5.5	27.2
Westcoast	172.2	250.7	248	15.7	6.4	25.6

† (86 percentile of January Maximum - 14 percentile of July Minimum)

then successive choices of variables were made that increased the discriminating power of the model. This process continued until the gain was no longer statistically significant. In all, twelve variables were selected in this stepwise fashion. These are listed in table 2.9.2 which also gives the group means for each variable. The means are interesting in their own right, for example, the Interior possesses the highest mean values for height and spirometry and Hobart possesses the lowest. The Northwest has the lowest value for productive cough and for its duration. Huon is characterised by low values for pneumonia, hives and wheezing and the highest values for infantile feeding difficulties, age of onset of productive cough, and deformities of the spine and thorax.

From these variables, discriminant functions were computed. The first three functions gave significant increases in discriminatory power and were retained for classificatory purposes; the remaining, insignificant functions were discarded. The structure of the functions is illustrated in table 2.9.3.

Function 1, which describes over forty-six per cent of the between group variance, is positively related to a history of pneumonia, the average duration of productive cough and the forced expiratory volume in half a second but is negatively related to vital capacity and maximum expiratory flow rate. This function is obviously describing a parameter of pulmonary health linked to pathological changes caused by pneumonia and extended bouts of bronchial infection.

Table 2.9.2

Group Means for Each Variable Entered in the Analysis

	Northwest	Hobart	Interior	Huon
Pneumonia	1.37	1.43	1.26	1.21
Hives	1.47	1.29	1.49	1.24
Age of Onset of Wheezing	2.50	2.41	2.40	2.95
Duration of Cough	1.55	2.55	2.49	2.09
Height	474.93	474.00	483.16	466.24
Forced Expiratory Volume $\frac{1}{2}$ sec.	1131.76	1058.50	1241.00	1180.07
Vital Capacity	1611.26	1520.23	1728.71	1621.57
Maximum Expiratory Flow Rate	1996.66	1732.95	2162.77	2082.48
Index of Wheeziness	1.00	0.99	1.00	0.98
Index of Productive Cough	1.45	1.76	1.89	1.71
Index of Upper Respiratory Infection	0.55	1.27	1.06	1.17
Index of Deformities	0.00	0.00	0.00	0.02

Table 2.9.3

Standardised Discriminant Function Coefficients*

	Function 1	Function 2	Function 3
Forced Expiratory Volume $\frac{1}{2}$ sec.	.90	+	.59
Maximum Expiratory Flow Rate	-.90	-.32	+
Vital Capacity	-.72	+	+
Average Duration of Cough	.52	-	.20
Height	.47	.53	.27
Upper Respiratory Infections	.26	-.58	-
Age of Onset of Wheezing	-	-.47	-
Spinal-Thoracic Deformity	-.26	-.41	+
Infant Feeding Difficulties	-.29	-.30	+
Index of Wheezing	+	.29	+
Incidence of Pneumonia	+	.22	-
Incidence of Hives	-	.34	.22
Index of Productive Cough	+	-	.50
Eigenvalue	0.317	0.227	0.137
Relative Percentage	46.5	33.3	20.2
Canonical Correlation	0.49	0.43	0.35
Wilks' Lambda	0.54	0.72	0.88
Chi-Square	109.79	60.10	23.23
Degrees of Freedom	39	24	11
Significance	< 0.001	<0.0001	0.016

* Coefficients less than 0.2 are indicated by + or -

Function 2 accounts for a further thirty-three per cent of the variance. It is characterised by positive relationships with hives and wheezing and negative relationships with age of onset, upper respiratory infections and thoracic deformities. This parameter is related to early-onset, allergic, wheezing of a mild nature.

Function 3 explains the remaining twenty per cent of the variance and is related to the history and presence of productive cough and the forced expiratory volume in half a second. This parameter is more difficult to describe as it loads on fewer variables but is probably related to bronchial asthma of a more severe, incapacitating nature than that described by function 2.

Classification

The next step was the production of a set of classification coefficients that would transform the raw variables for each case into probabilities of membership for each group. Each case is thus allocated to the group for which it has the highest probability. For the originally "grouped" cases, the proportion of correct allocations is calculated to give an estimate of the predictive quality of the model. The ungrouped cases' allocation are then examined to see how accurately a case of unknown group affiliation can be located. The results are given in table 2.9.4. Just under fifty per cent of the grouped cases were correctly assigned. This proportion is about twice the proportion expected purely by chance (25 per cent).

By examining figure 2.9.2 one can understand the pattern of misclassification. Here, the sample cases are plotted in two of

Table 2.9.4

Prediction Results

Grouped Cases	No. of cases	Allocated Groups			
		Northwest	Hobart	Interior	Huon
Northwest	45	21 46.7%	9 20.0%	8 17.8%	7 15.6%
Hobart	91	10 11.0%	45 49.5%	19 20.9%	17 18.7%
Interior	39	4 10.3%	6 15.4%	20 51.3%	9 23.1%
Huon	47	6 12.8%	9 19.1%	9 19.1%	23 48.9%
Ungrouped Cases	1165	255 21.9%	321 27.6%	287 24.6%	302 25.9%
TOTAL	1387	296 21.3%	390 28.1%	343 24.7%	358 25.8%

Percent of "Grouped" Cases Correctly Classified : 49.1%

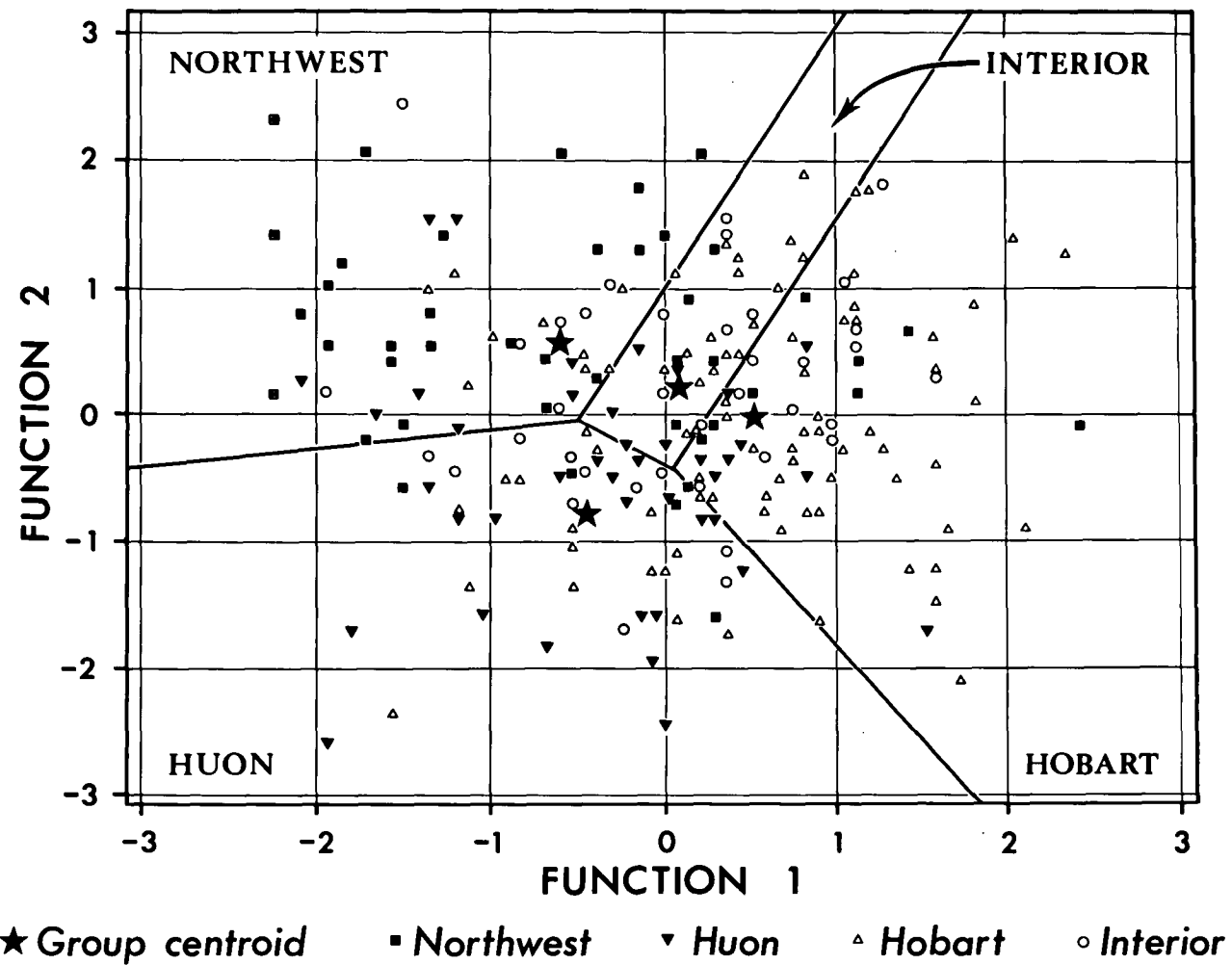


Figure 2.9.2 Location of the Geographic Samples in Discriminant Space

the variable dimensions of discriminant space. Each case group membership is indicated by a different symbol and the territories of the different types have been marked with lines that bisect the distances between the group centroids. The overlap is immediately obvious; the Interior type's territory has considerable invasion by cases of other types. With a heterogeneous complaint like asthma this is really not too surprising. Many cases will tend towards the average and cluster near the centroids, only the extremes in each area provide the variance for discrimination.

Although the three discriminant functions were found to predict group membership correctly for fifty per cent of the cases with known affiliation, ungrouped cases had also been classified. It remained to be discovered whether these extra cases would enhance the prediction results and also whether the grouped and ungrouped cases, taken together, could demonstrate geographic patterns of statistical significance.

Results and Discussion

Figure 2.9.3 presents the results of the spatial clustering. The probability maps obtained are similar to those of the Choynowski/McGlashan⁷ method but are based upon the binomial rather than the Poisson distribution. All of the shaded areas shown are significant at $p \leq 0.01$, unless otherwise stated. In addition, an attempt has been made to indicate "high spots" within the areas of significance. With large numbers it is often possible to obtain a significant deviation with a remarkably small increase in proportion. Each area type has an intense

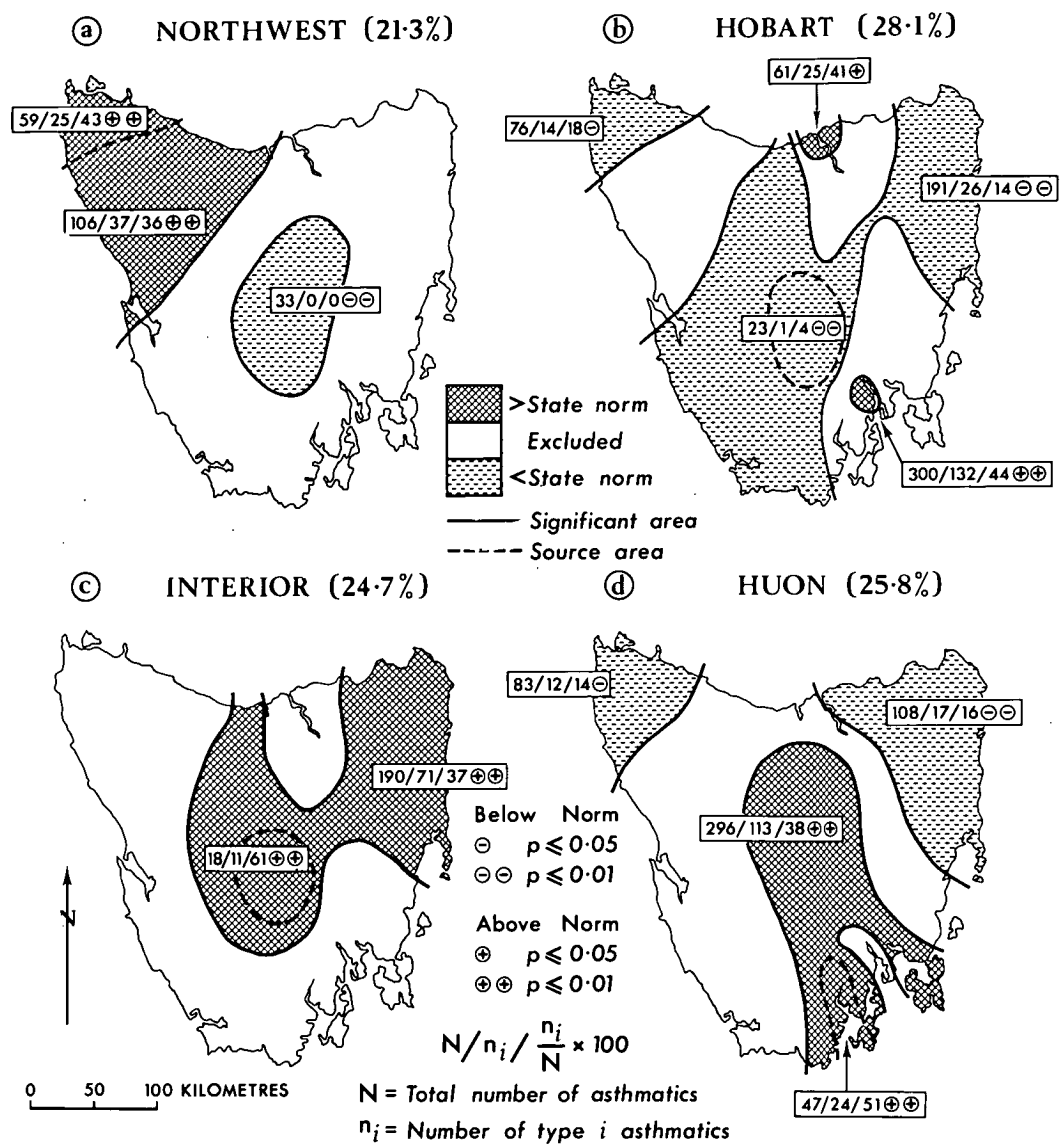


Figure 2.9.3 The Spatial Distribution of Members of Each Group

local effect surrounded by an area of overall increased prevalence that is statistically significant. The blank areas on the map are excluded areas that are close to the norm in prevalence.

When figures 2.9.1 and 2.9.3 are compared it is possible to assess the spatial persistence of the types. The constituent members of the type groups were from quite small geographic areas; however, all types demonstrate in the maps much larger areas of above average prevalence. The source area for each type usually has the highest concentration of that type. This core locality is surrounded by an area of lowered prevalence that remains significantly higher than the norm. A distance decay gradient is observed about these core areas but is disturbed by the population's affiliation to other types.

The Northwest type has a state prevalence of about twenty-one per cent (Figure 2.9.3.a). In its source area its prevalence is twice the norm at forty-three per cent. When the peripheral areas are added the prevalence drops to thirty-six per cent which is still significantly greater than the norm at $p \leq 0.01$. Northwest type asthma is absent from the central area of the state, $p \leq 0.01$, as this area is dominated by Interior and Huon types.

Hobart type asthma (Figure 2.9.3.b) was represented by the school populations in the core of the industrial manufacturing areas of the city. With a state prevalence of twenty-eight per cent the only high areas are the western suburbs of Hobart, forty-four per cent, $p \leq 0.01$, and an area around Georgetown at the mouth of the Tamar estuary, $p \leq 0.05$. Other urban and suburban areas were

close to the state norm. Similarly to Northwest type, Hobart asthma was absent from the central area and most of the rural districts, $p \leq 0.01$. The far Northwest area was also low, $p \leq 0.05$.

Interior type (Figure 2.9.3.c) accounted for nearly twenty-five per cent of the cases. Its high prevalence area closely parallels Hobart type's low prevalence area. The source area contains sixty-one per cent of its own type; its larger region, thirty-seven per cent, $p \leq 0.01$. The only significantly low area discovered was the industrial area of Hobart, sixteen per cent, $p \leq 0.05$.

Huon type (Figure 2.9.3.d) had a prevalence of fifty-one per cent in the Huon area, twice the norm. Its wider domain included the Interior source area and gained a prevalence of thirty-eight per cent. The suburbs of Hobart, possessing only close to average proportions, formed an enclave within this area. The Northeast sector of the state, an area of high Interior type prevalence, was significantly low for Huon type, $p \leq 0.01$.

By examining table 2.9.1 and the additional detailed information upon climatic averages in table 2.9.5 one can begin to identify the links between climatic environments and the discriminant groups. When extremes in the spatial patterns in figure 2.9.3 correspond to extremes in the climatic data, evidence for environmental interaction is strongly supported. Actually, the maps suggest two pairs of contrasting dimensions. Hobart contrasts with Interior and Northwest with Huon.

The first pair, Hobart and Interior, has obvious differences in environment; they measure the opposite ends of the rural-

Table 2.9.5

Climatic Averages for Representative Stations

	Northwest	Huon	Hobart	Interior
<i>Annual Averages</i>				
9 a.m. Temperature	13.0	11.6	12.1	10.3
3 p.m. Temperature	15.4	15.3	15.3	14.9
Frosts, light ($\leq 2^{\circ}\text{C}$)	7	n.a.	13	138
Frosts, heavy ($\leq 0^{\circ}\text{C}$)	1	n.a.	1	87
<i>Monthly Averages</i>				
January				
Maximum Temperature	20.9	21.3	21.8	22.8
Minimum Temperature	12.5	8.8	11.9	7.6
Temperature Range	8.4	12.5	9.9	15.2
July				
Maximum Temperature	12.5	11.5	11.6	10.8
Minimum Temperature	6.1	0.0	4.5	-0.3
Temperature Range	6.4	11.5	7.1	11.2

urban continuum. The Hobart group comes from the polluted, industrial parts of the city; the Interior group from the clean, open bushlands, 300 metres higher and 100 kilometres inland. When the group means are compared, Hobart's spirometry values are the lowest and Interior's are the highest. The other notable differences in symptoms are the increased weighting of upper respiratory infection and the increased incidence of pneumonia in the Hobart type. The extremes in spirometry may well be related to a beneficial effect of altitude in the Interior and a harmful effect of low level pollution in Hobart. Sulphur dioxide, in particular, has a marked relationship to increased airways resistance. Interior residents are subjected also to a greater range in diurnal and seasonal temperatures. Although only 50 km from the nearest coast, topography and altitude produce a degree of continentality in the area. This can be seen from the number of frost days, the January average temperatures, the annual range and the low precipitation. The differentiation of these two areas is probably related to the first discriminant function which loaded highly on the spirometry variables.

The Northwest and Huon types are both from rurally-based samples; one in the far North of the state, the other in the far South. In comparison to the Northwest, the Huon type loads highly on age of onset, the average length of attack of productive coughing, the history of upper respiratory infection and thoracic deformities. It loads negatively on hives and height. The Northwest has slightly lower spirometry values, a low value for upper respiratory infections and the lowest value for average

length of duration for productive cough. Age of onset is about six months earlier, on average, in the Northwest. The separation of these two areas is probably related to the second and third discriminant functions. Function 2 reflects the intermittent, short duration, early onset, allergic wheezing of the Northwest while Function 3 reflects the extended bouts of crippling bronchial asthma of later childhood. This would appear to be involved with differences in temperature. Although the Huon area is by no means continental, it has the most southerly latitude of the groups. Its climatic variables from table 2.9.5 are closest to those of the Interior. The average July minimum of 0°C is 5°C lower than the Northwest and emphasises the increased probability of days of frost. The Northwest, although not the warmest of the areas, does experience the smallest range of temperature and the highest July minimum. The moderating effect of the ocean is important in this location. In the Huon area, latitude and cold air drainage in Winter counteract the maritime influence.

More evidence is obtained from the Huon map when it is compared to that of the Interior. One notices that Huon type's larger area of significantly high prevalence embraces the central portion of the state - the Interior source area. If the suggestion of low temperature being culpable for Huon type asthma is correct, it would be expected to show this overlap with the cooler Interior environment. Further, the Northwest type "warm environment asthma" is conspicuous by its absence from the Interior source area.

Conclusion

The discriminant and cartographic analyses have thus highlighted three environmental and three asthmatic health dimensions that appear to interact with each other to produce the broad patterns of asthma morbidity in Tasmania. The major climatic parameter would be an aspect of temperature probably the prolonged cold periods experienced in particular environments acting in conjunction with the strong fluctuation in temperature common to continental areas. High altitude may also be indirectly involved in asthma pathogenesis in that reduced pressure, reduced partial pressure of oxygen and reduced pollution may have beneficial pulmonary effects. The third environmental element is the price paid for living in urban environments: the reduced pulmonary efficiency caused by domestic, vehicular and industrial pollution. The inferred relationships between the discriminant functions and the environment dimensions are summarised in table 2.9.6.

To a large extent this analysis has been exploratory. The relationships outlined above are fairly conjectural. Enough evidence has been produced, however, to structure hypotheses that can be tested in future work. Whatever further research determines to be the exact relationships between all of the factors involved, it has been demonstrated that with one chronic complaint, asthma, geographic types can be successfully distinguished from one another.

In this four group example the highest level of predictive accuracy was sixty-one per cent (in the source area

TABLE 2.9.6

Inferred Relationships between Discriminant Functions and Environmental Variables

	Function 1 Pulmonary Function	Function 2 Mild Wheezing	Function 3 Bronchial Infection
Temperature	-	+	-
Altitude	+	-	+
Pollution	-	-	+

of the Interior type of asthma). Imperfect predictive ability is, in part, due to the heterogeneity of the syndrome and, in part, due to migration noise that was not controlled for in the data. With more stringent controls and increased selectivity in group membership prediction can be made far more successful. For example, all of the individuals located by primary school were assumed to have spent their maximum period of residence in that location. This assumption is unrealistic for the Hobart area but is more reasonable in isolated, rural localities where out-migration dominates.

Another method of increasing the resolution of the analysis would be to take different sub-populations of wheezers separately. In the present analysis the entire population was included. Individuals ranged from those who had wheezed once to those with continuous symptoms. It can be hypothesised that any environmental effects would have had differential impacts on these and other subtypes. This option, however, was impossible to follow up because of the smaller numbers involved when the population was subdivided.

2.10 Summary

This section has been devoted to the analysis of a total geographic survey of the 1961 Tasmanian birth cohort. From different perspectives and in many ways, investigations have proceeded from those concerned with single variables to the examination of multivariate constructs. Each analysis has concluded with probability mapping and the detection of significant spatial clustering. Areas delineated by this method have indicated the locations with high potential for asthma-environment exploration. In these extreme areas of prevalence it was assumed that any causal/irritant factor(s) were acting with maximum effect, and therefore were more easily observed.

Because of the many approaches adopted to the same set of data and its constituent variables, the cartographic and tabular products of analysis were numerous and their relationships complex. To assist interpretation, and to condense the evidence presented to manageable proportions, the whole body of findings is here drawn together then centred around a few common aspects of asthma morbidity and the physical environment. How do the maps generally agree upon morbid and healthy environments and, specifically, how do spatial patterns identify with patterns due to urbanisation, physiography or climate?

Urban-Industrial Patterns

Generally, urban areas because of their larger numbers, increased environmental heterogeneity and migratory population, tended toward the average and reflected the state norms for

many of the variables considered. In a large number of maps Hobart and Launceston, and to a lesser extent Burnie and Devonport, fell into excluded areas because of their average characteristics. The city of Hobart, however, did enjoy a slight surplus of persons whose attacks were of short duration (figure 2.3.4.a). Urban effects were first noticed in the spirometry maps (figures 2.5.2 to 2.5.5). In female spirometry maps the urban areas are close to average with the exception of M.E.F.R. scores greater than 0.5 standard deviations. Hobart was deficient in its proportion of healthy female M.E.F.R. The spirometry distributions of males were much more markedly affected by urbanisation. The Hobart area and particularly the city of Glenorchy were characterised by significant surpluses of below average scores and deficits of healthy values. This could be linked to the decreased atmospheric quality in the urban environment and the industrial pollution from factories in the northern suburbs.

The next urban concentration was noted in the spatial distributions of individuals from the most severely-affected group described by the cluster analysis (figures 2.8.3 to 2.8.5). A significant prevalence of this type of asthmatic was found in the Launceston-Tamar region. This was found to be true for both sexes although their patterns were slightly different. Hobart, conversely, was prominent for its excess of females belonging to the remission cluster.

An urban dimension was built in to the discriminant analysis by sampling children from a spatially-restricted cluster

of schools in the industrialised suburbs of North Hobart and Glenorchy. This group of children had the lowest mean scores for spirometry of all the groups entered in the analysis. The spatial pattern of the population classified as most similar to them was illustrated in figure 2.9.3.b. Concentrations were limited to the West-shore suburbs of Hobart and the industrial parts of the Tamar estuary. Launceston and the towns of the Northwest Coast possessed average proportions and the predominantly rural parts of the state contained very few of this group.

The remaining urban feature that was observed was the delayed age of onset in the Hobart area. This was indicated by the high numbers of new female cases found between 1968 and 1974 (figures 2.6.1.b and d.) in this area; and the above-average numbers of new male cases found in the wider ecumene generally. The urban epidemic of onset after the seventh year followed from the progression of onset in earlier years demonstrated in figure 2.3.1. One hypothesis concerning this delay in onset is that any urban heat island effect may possibly help to moderate thermal stress. This may be of importance to some asthmatics; urban residence in combination with clean, efficient household heating and the avoidance of thermal stress could form the basis of primary preventive management.

Maritime-Continental Patterns

The significance of differences between coastal and inland areas was difficult to assess because of the small numbers of inland residents. The coastal populations also tended to be highly urban and any positive maritime influences were masked by

the effects of urban residence discussed earlier. Inland residents possessed non-urban lifestyles and lived, on average, at higher altitudes than those at the coast. A comparison of the two populations therefore had to take into account at least altitudinal, urban and maritime effects.

Because of the heterogeneity of the coastal environments, a universal maritime pattern was not evident. Certain coastal areas, however, did show some consistency in their patterns. The East and Southeast coastal areas had a higher average duration of episode (figure 2.3.4.c), incidence of pneumonia (figure 2.2.2.c) and total number of episodes (figure 2.3.5.c); and a low proportion of healthy F.E.V._{1/2} sec. (figure 2.5.4.b) in males, and low scores in males' spirometry generally (figures 2.7.2.a and b).

The Northwest Coast was different to the Southeast in almost every respect. West of Burnie there was a significant increase in remission of symptoms in females (figure 2.6.1.c), a surplus of early onset cases (figure 2.3.1.a) and a high number of single attacks (figure 2.3.5.a). Over the entire area the prevalence of hayfever was low but that of flexural eczema was high (figures 2.2.2.a and d). In contrast to figure 2.6.1.c, the cluster of females enjoying remission of symptoms was underrepresented (figure 2.8.5.c). This was probably due to the effect of including the industrial towns of Burnie and Devonport.

The Northwest was one of the four areas entered in the discriminant analysis (figure 2.9.3.a). The samples used for this analysis were from the rural-coastal population residing West

of Burnie. On average, their experience was of early-onset, mild wheezing. This area was much influenced by its maritime location, mild winters and low diurnal-range in temperature.

Very few strictly inland clusters were observed. Several patterns grouped the central parts of the state and other rural, including coastal, areas but few were isolated central clusters. Exceptions were figure 2.2.1.c that illustrated a deficit of total female wheezer prevalence; figure 2.8.4.a which demonstrated a surplus of mild male wheezers and figure 2.7.4.d that showed a large number of female remissions. The latter actually included a small coastal stretch in its area as did several other maps, for example, figure 2.3.2 of late-onset remissions, figure 2.2.2.c of low pneumonia prevalence and figure 2.2.2.d of high flexural eczema prevalence.

Again, the discriminant analysis helped to piece the picture together. The sample of asthmatics chosen to represent the inland area (Interior) was solely from rural settlements in the central parts of the state. Here, the diurnal range of temperature was widest and any maritime effect at its weakest. The highest prevalence of this type was in its sample area but significantly high proportions were found in a large area that included most of the high country in the Northeast quadrant of the state. The group's characteristics were assumed to represent the comparatively healthy environmental interaction supplied by rural, higher-altitude locations.

Latitude and Temperature

Latitude, altitude and temperature are closely inter-related. The latter variable is universally related to the other two and, in the spatial domain, altitudinal changes can mask any latitudinal gradients. The most pronounced latitudinal pattern seen in the data was that of the wheezers without cough. Figures 2.2.1.d, 2.7.3.b, 2.8.3.e and 2.8.4.e all showed an increased proportion of non-bronchial wheezers (defined by different methods) in the northern parts of the state and a lack of them in the South. Urban areas were close to average. The most obvious interpretation of such a pattern was that some aspect of temperature was involved in the pathogenic process. This observation was reinforced, again, by the discriminant analysis. One group (Huon) was a sample from the southernmost inhabited parts of Tasmania. Figure 2.9.3.d showed that this group type was significantly under-represented in the northern parts of the state; but that significantly high numbers were found in the higher-altitude, cooler, interior regions. Temperature, therefore, was seen to be a common parameter between high-latitude and high-altitude environment asthma. The two areas were not entirely congruent and an additional factor, possibly ionisation, was thought to be implicated in the difference between these two groups.

To recapitulate, several significant spatial clusters have been demonstrated repeatedly after many different analyses of the data. Reproducibility of patterns in this way has given weight to the meaningfulness of the spatial clusters produced.

Some of the more robust clusterings were observed to parallel the spatial fluctuations of particular physical processes. This led to the association of asthma morbidity with urbanisation, pollution, maritime effects, continental effects, latitude and altitude. A common principle to all of these was some dimension of temperature.

Because of the last observation and the many different clues from the maps and the literature a biometeorological approach now becomes mandatory. Morbidity and its relationship to the physico-chemical nature of the atmosphere can only be modelled satisfactorily from a dynamic point of view. Static cross-sectional analyses have given the broad spatial scale of interaction; the elucidation of more precise relationships will require temporal insights into the stimulus-response characteristics of asthmatics in a given climatic regime.

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SECTION 3

HOSPITAL MORBIDITY STATISTICS

3.1 Introduction

Section two established the existence of significant geographic variation in asthma morbidity within Tasmania. Subsequent interpretation of regional variation highlighted the parallels between particular morbidity clusters and the broad spatial expression of the processes of urbanisation and climatic gradation. Changes in climatic regime due to latitude and altitude were particularly evident. The underlying element common to urbanisation, altitude and latitude was thought to be temperature. The implication of some dimension of temperature, furthermore, conformed with current biometeorological theories upon asthma and the effect of asthmospheric cooling upon morbidity.¹

The next step was to obtain historical data upon the temporal variations in numbers of attacks preferably for the total and the several parts of the state. This type of information was necessary for the detection of significant variations over time in testing the second null hypothesis. This was concerned with fluctuations in morbidity time series for groups of asthmatics defined by different criteria one of which

was residential location. Also, it was hoped to obtain morbidity records on a daily basis so that the relationships between morbidity and daily meteorological variables could be investigated in detail. It was intended to extend this investigation to several of the extreme areas delineated in section two and thus detect any differences between stimulus-response, meteorotropic, mechanisms acting in different geographic locations.

Hospital morbidity statistics, in the form of daily admissions to public hospitals for asthma as the principal condition, have been commonly used as an index of acute morbidity in studies of asthma and its interaction with the atmospheric environment. Many of these investigations were reviewed earlier. Tasmania has been comparatively neglected by this type of research activity because of its much smaller total and capital city populations: and Australian studies, generally, have been restricted to the correlation of attendance rates at major city hospitals with meteorological data from the nearest weather station. The morbidity data used in this type of analysis usually have not been adjusted for age, sex or residence and yet this crude index of asthmatic distress has often produced striking correlations with weather elements. An examination of hospital morbidity data was included here for comparative purposes; for the detection of spatiotemporal variation and to aid hypothesis generation with respect to morbidity-environment interaction. Indeed, the uses of this type of data were thought

to be far greater than has been considered hitherto particularly in reference to age, sex and geographic sub-populations.

Hospital morbidity data, however, are not without limitation. Admissions for asthma attacks are particularly likely to be affected by social factors. Persons of low socioeconomic status will probably be over-represented in contrast to those of high socioeconomic status who will tend to be associated with private practice. This relationship may, however, be overridden in an emergency. Superimposed upon this social pattern will be a distance-decay effect. Persons living far from hospital facilities may tend to cope with or tolerate a greater degree of morbidity than those sufferers who live close to a hospital. They may delay in initiating a high-speed car journey in the hope that the attack will subside. After a long delay before the decision to seek help is made, half-way to the hospital many often experience relief of symptoms. Asthmatics who live at the fringes of hospital catchment areas may rely to a larger extent upon general practitioners and over-the-counter remedies than their city counterparts. For the socially and spatially (especially transportation) disadvantaged the access to any form of care may be severely limited. Lastly, superimposed upon the socio-spatial patterns of access are temporal fluctuations in access and utilisation.

A so-called "weekend-effect" may be built into the time series of morbidity. There are at least three mechanisms that can contribute to this pattern of morbidity. The first is the reduced access to general practitioners at the weekend. This

would help to account for any increase on Saturdays or Sundays. Second, sporting fixtures and other unusually strenuous forms of exercise tend to cluster at weekends and exercise-induced asthma may be more common at this time. A third, a hypochondriacal effect based on childhood aversion to school and an adult aversion to work might contribute to increased attacks on Fridays and Mondays. With all these deficiencies kept in mind, the data are used here for three reasons: they are available on a state-wide basis, they are comparable to other studies and, assuming all errors are consistent over time, they represent a fair index of acute attacks.

Admissions data were obtained from the Australian Bureau of Statistics for all admissions to public hospitals in Tasmania from 1972 to 1977 where the principal condition treated was asthma. For each admission five pieces of data were made available; the date of admission, sex, age, local-government-area of usual residence and the length of stay. For 1977 only, additional information was obtained upon multiple admissions. It was thought that persons suffering from a chronic complaint would have a tendency to make more than one visit a year and that such multiple admissions could seriously affect the calculation of morbidity rates especially in areas of low population. The frequency of admissions was used to estimate the community prevalence of the hospital-oriented asthmatics. Again keeping the limitations of the data in mind, it was possible to select sub-populations from the data in order to compare differences due to sex, age, residence and their various combinations.

3.2 Public Hospital Admissions for Asthma: Tasmania 1972-77

Temporal Patterns

In all, there were 3746 admissions for asthma during the six year period. The annual average number of admissions was 624 but this varied from 511 in 1977 (-18 per cent) to 721 in 1973 (+16 per cent). This annual variation was illustrated in figure 3.2.1. Although the annual totals seemed to describe a downward trend or some type of cyclical behaviour, the sample of years was too small to establish any such effect. Figure 3.2.2.a. was much more interesting. Here, among the monthly averages, the month of March was prominent. It possessed, on average, 11.6 per cent of total admissions. This was half as much again as the average monthly proportion of 8.3 per cent. March, situated at the beginning of the Tasmanian Autumn, paralleled other studies of asthma episode seasonality. This effect was seen to be more marked in males than females. An examination of figures 3.2.2.b and 3.2.2.c showed that thirteen per cent of male admissions were in March compared to less than ten per cent of female admissions. Females, instead, demonstrated a slight increase in October admissions, just under ten per cent. Male admissions were lowest in June, 6.5 per cent, and only slightly higher in January, 6.8 per cent. Female admissions were lowest in September, 6.6 per cent. In males the Autumn peak was dominant and was followed by a low trough in Winter and a gradual rise in Spring that dropped again in Summer. In females

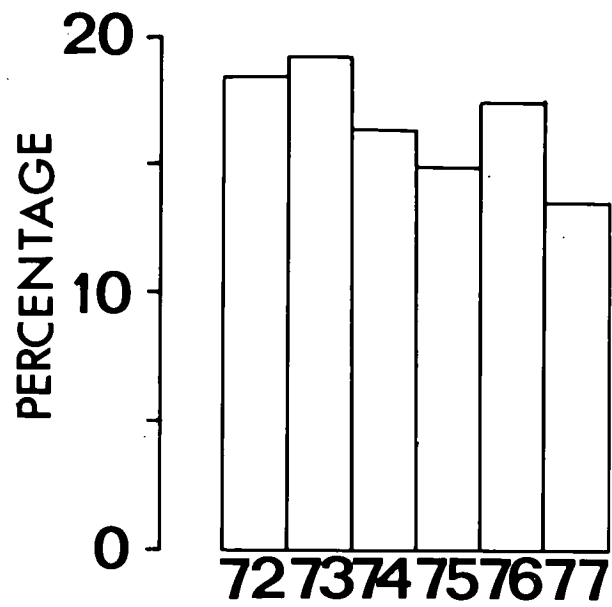


Figure 3.2.1 Annual Admissions to Hospital in Tasmania for Asthma 1972-1977.

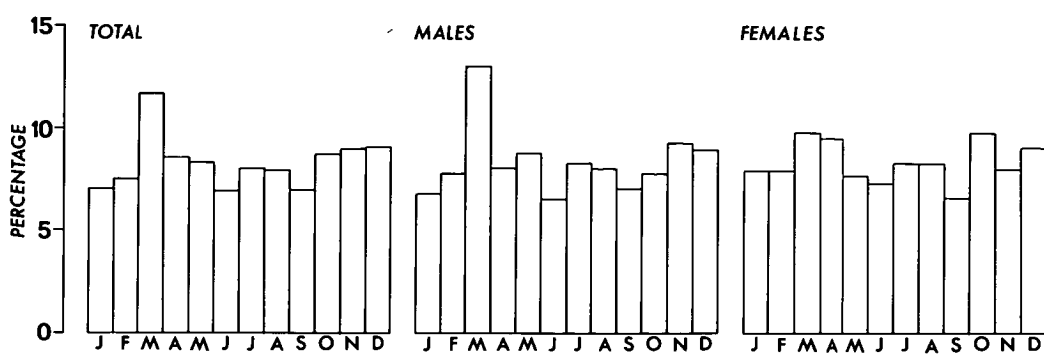


Figure 3.2.2 Percentage Distribution of Monthly Admissions to Hospital in Tasmania 1972-1977.

the Autumn and Spring peaks were equivalent and the Winter and Summer levels close to average.

As was discussed earlier, it was suspected that a weekend effect might influence the weekly pattern of attacks. To observe if this was the case all the admissions for one year, 1977, were coded for day of the week and a histogram was produced of the daily Tasmanian frequencies (figure 3.2.3). Although on average, Friday and Saturday enjoyed slightly lower admission rates and Sunday and Monday suffered slightly higher ones, the daily absolute sums differed insignificantly from the mean, (χ^2 of 6.28 with six degrees of freedom). It was decided, therefore, that any weekend effect was negligible and could be ignored as a cyclical factor.

Age/Sex Differences

One difference due to sex has already been noted; the seasonal peaking of attacks in March in the male population. Of the 3746 cases, 2155 (57.5 per cent) were males. The average sex ratio of attacks was 135.5 males/100 females. This ratio was found to vary enormously with age (table 3.2.1). In the first decade of age, males exceeded females in admissions by a ratio of 214:100. This quickly dropped to a ratio of 37 males/100 females in the third decade. It was not until the sixth decade that male admissions again exceeded the females'. In the very young, and in the aged, male morbidity was dominant. In the teenage years female admissions grew in proportion, peaked during the twenties and thirties and were diminished by an increasing proportion of admissions for males over the age of fifty.

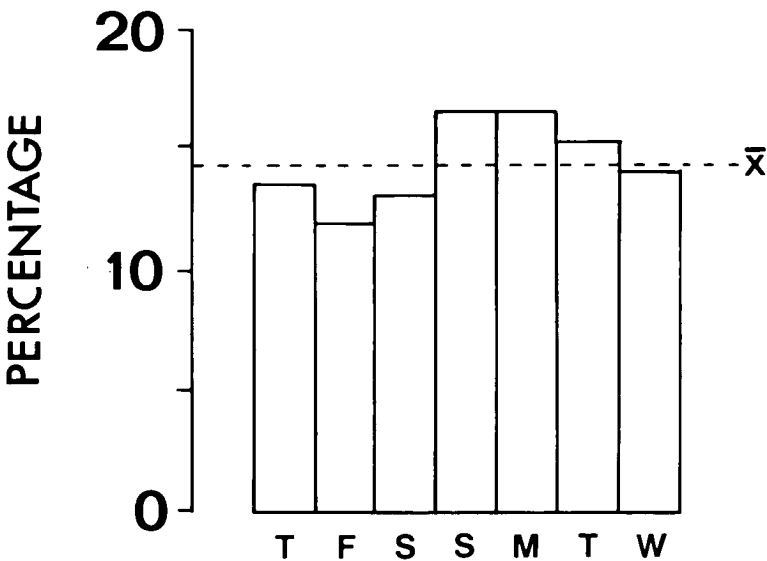


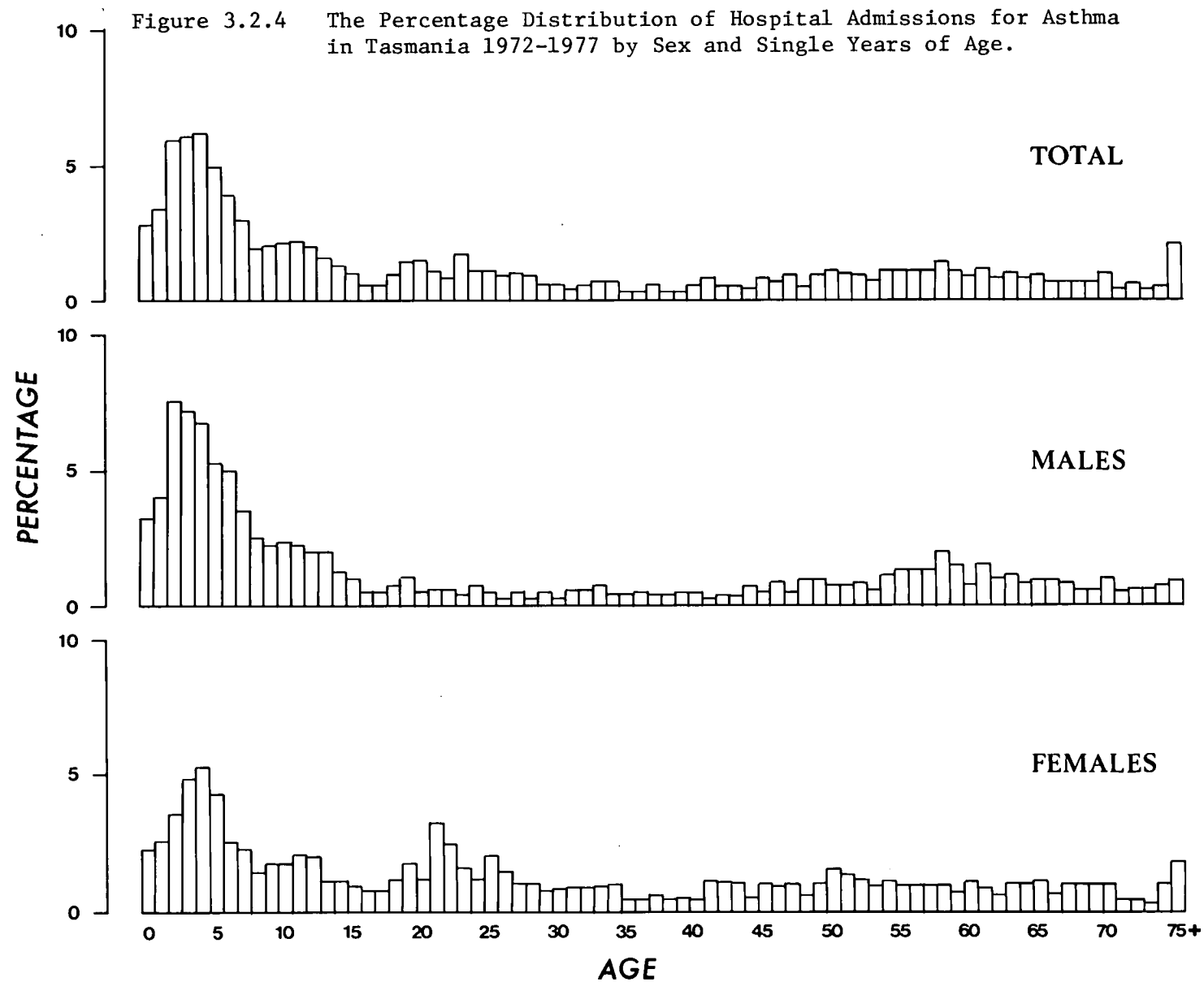
Figure 3.2.3 Percentage Distribution of Hospital Admissions for Asthma in Tasmania 1972-1977 by Day of Week.

TABLE 3.2.1
Hospital Morbidity by Age and Sex 1972-77

Age Group	Males		Females		Total		Sex Ratio
	N	%	N	%	N	%	
0 - 10	1076	(49.9)	502	(31.6)	1578	(42.1)	214.3
11 - 20	266	(12.3)	223	(14.0)	489	(13.1)	119.3
21 - 30	100	(4.6)	271	(17.0)	371	(9.9)	36.9
31 - 40	91	(4.2)	101	(6.3)	192	(5.1)	90.1
41 - 50	120	(5.6)	143	(8.9)	263	(7.0)	83.9
51 - 60	234	(10.9)	149	(9.4)	383	(10.2)	157.1
> 60	268	(12.4)	202	(12.7)	470	(12.5)	132.7
TOTALS	2155	(100%)	1591	(100%)	3746	(100%)	135.5

Figure 3.2.4 illustrates in the form of histograms, the occurrence of admissions of each sex for single years of age. The first histogram (3.2.4.a) takes both sexes together. Its multi-modal structure is obvious; a peak of admissions during the first seven years, another smaller one during the next seven, yet another during the twenties, a trough in the thirties and early forties and a rather flattened but symmetrical distribution, peaking in the late fifties and early sixties. When the sexes are examined separately the differences shown in table 3.2.1, are given more substance. The first two modes are proportionally greater for males, the third mode is dominated by females and the last mode, again is better defined in the male histogram. Another approach is taken in figure 3.2.5; here the cumulative percentages of admissions for each sex are plotted on the same graph. If the quartile ages are examined, it is observed that in males the first twenty-five per cent of admissions are aged three or under, the first fifty per cent are aged ten or under and the first seventy-five per cent are aged forty or under. In females the ages are six, twenty-three and forty-seven years respectively. By the end of the third quartile, in the late forties, the sexes seem to reach an equilibrium in accumulated admissions.

So far, the underlying age-sex structure has not been taken into account in the generation of these admission statistics. Figure 3.2.6 was based upon average, annual, age-sex specific, admission rates per hundred thousand population. These were calculated by taking the annual average for each five-year stratum



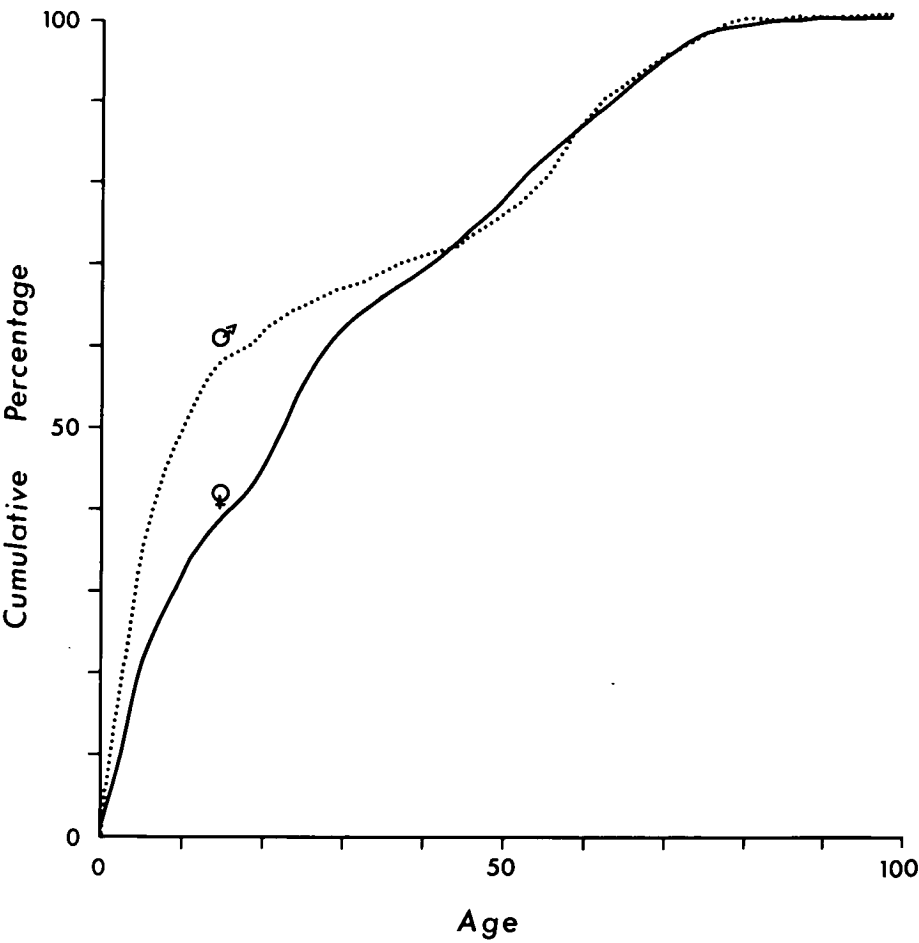


Figure 3.2.5 Cumulative Percentage of Hospital Admissions for Asthma in Tasmania 1972-1977 by Sex.

by sex, dividing it by the average state population for that stratum and multiplying it by 100,000. The average state population was calculated from the 1971 and 1976 censuses. The graph for the separate sexes removed any visual bias due to unequal population distribution. The basic pattern was similar to that already described, but it highlighted especially the female peak in the twenties and the male peak in the fifties and sixties.

The surplus of male admissions in the early years reflects the generally increased susceptibility of this age-sex stratum to illnesses and health hazards. The increasing dominance of females throughout puberty and early middle-age may be linked with hormonal changes, different physiological responses to environmental stresses such as cold or may reflect a poorer prognosis for females. Also, many female asthmatics only have asthma during pregnancy. This would help to explain the increased incidence in the prime childbearing years. The increase in male admissions during late middle-age could be related to the long term effects of occupational exposure to respiratory irritants or to self-induced pathologies from smoking habits. Women here would be under-represented because of their more limited occupational exposure and smoking practices.

Age-Sex and Seasonality

Because of different susceptibilities, exposures and stresses at the various life-stages, it was hypothesised that the different age-sex strata would demonstrate dissimilar seasonal

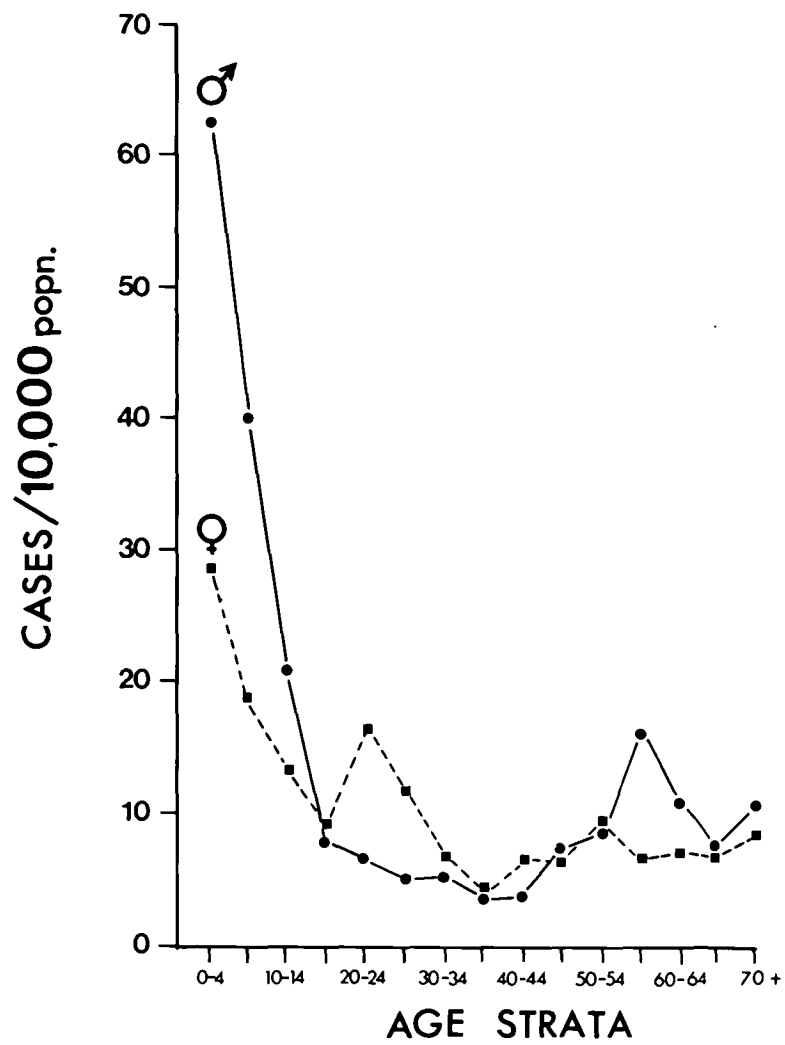


Figure 3.2.6 Average Annual Age-Sex Specific Hospital Admission Rates: Tasmania 1972-1977.

patterns. To investigate this idea, average monthly admissions were calculated for each sex by ten-year, age strata. To assist comparison, the absolute admission frequencies were converted to percentages of total admissions and the results plotted as a series of histograms (figure 3.2.7). Changing seasonal patterns can be viewed either longitudinally for each sex or as a series of pairs contrasting the sexes for each age-group.

The male seasonality pattern is generally more striking than the female one. In the first decade of life the early Autumn peak is strongest; this persists until the fifth decade when the late Autumn-Winter peak becomes dominant. A Spring peak is not evident until the thirties, and it does not persist past this time. An early Summer peak can be seen in the twenties and a late Summer peak in the thirties. From the end of the third decade onwards Winter assumes a greater importance; the forties have an especially strong concentration of Winter admissions.

In the first ten years of life the female Autumn peak is much less than the male peak. This is offset by a late-Spring early-Summer peak. The patterns for the teens are not particularly strong. There are minor peaks in April and December in female attacks contrasting the March-May peaks. Both sexes demonstrate Autumn peaks in the twenties age groups. Males have an additional Spring-Summer excess probably indicating an increased susceptibility to pollenosis at this time of year. Females in the thirties are characterised by a large peak in

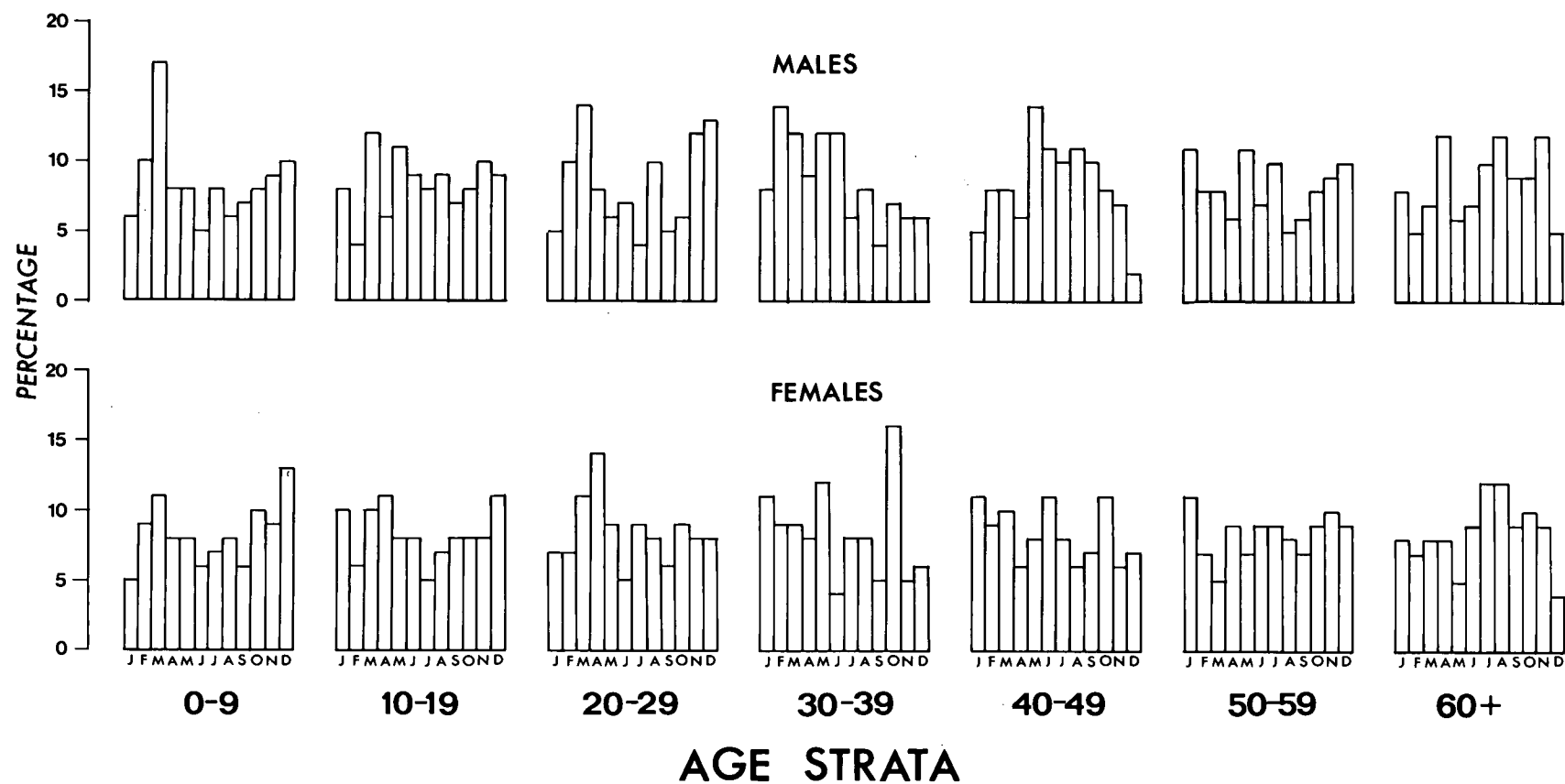


Figure 3.2.7 Seasonality of Hospital Admissions for Asthma in Tasmania 1972-1977 by Age and Sex.

October and smaller ones in January and May. Males in this age group have their admissions concentrated in Autumn-Winter. The female patterns for later years are weak. Any concentration is seen in Summer or Spring. This contrasts with the Winter dominance in males of later years.

Prevalence

The individual datum used here was a single admission to hospital. Each admission was unable to be linked to a patient identity number for reasons of confidentiality. Without such linkage it was impossible to know or estimate the size of the morbid population, 600 or so admissions a year could be generated by as many as 600 individuals or as few as a hundred. Fortunately, it was possible to obtain adequate information from the Australian Bureau of Statistics for one year, 1977. Multiple admissions by the same individuals were sorted on to a computer printout without any patient identifiers and the admissions belonging to the same individuals were clerically bracketted. In this way the numbers of single admissions could be counted separately from the number of two, three, and four admissions during 1977. Table 3.2.2 gives a breakdown of these statistics.

From the table it can be seen that the morbid population was about eighty-two per cent of the number of admissions. The 511 cases in 1977 were due to the illnesses of 417 individuals. Of these, 82.5 per cent paid single visits to hospital; 17.5 per cent were admitted more than once and only 3.8 per cent more than twice. In the calculation of prevalence from the morbidity data,

TABLE 3.2.2
Admissions to Public Hospitals in 1977

Frequency	Persons		Admissions	
	N	%	N	%
1	344	82.5	344	67.3
2	57	13.7	114	22.3
3	11	2.6	33	6.5
4	5	1.2	20	3.9
Total	417	(100)	511	(100)
% of Admission	(81.6)		(100)	

multiplication by 0.816 gave a fair estimate of the crude prevalence. If the average admission rate for the state was 624 per annum the average crude prevalence would have been $624 \times 0.816 = 509$ persons, or, about 130/100,000 population. The asthmatic population was, of course, much larger than this. Many asthmatics, due either to drug therapy or location or access to alternative care, did not appear in public hospital admission statistics. The figures were, however, useful in their own right both as an index of need and as an index of morbidity in a loosely-defined but presumably stable population of unknown bias. Figure 3.2.8 illustrated where the asthmatics who were admitted to the hospital during 1972-77 normally resided by local government area. Both absolute figures and rates per 100,000 population have been indicated.

Admission rates ranged from twenty to five hundred and twenty five in one hundred thousand. Two large areas of high rates stood out. The first was the combined West Coast and extreme North West corner of the State. The second was a central area of the East Coast extending inland to south of the Tamar estuary. The three major urban centres that contained the largest hospitals were surrounded by large areas with case ratios close to the norm. This was to be expected as a function of their size. Areas of low admissions were found in four locations; the South Midlands, the far South, the inland Northwest and to the West of Launceston. The low rate to the West of Launceston may have been due to residential miscoding. Many of the city's suburbs have encroached upon this area and local residents

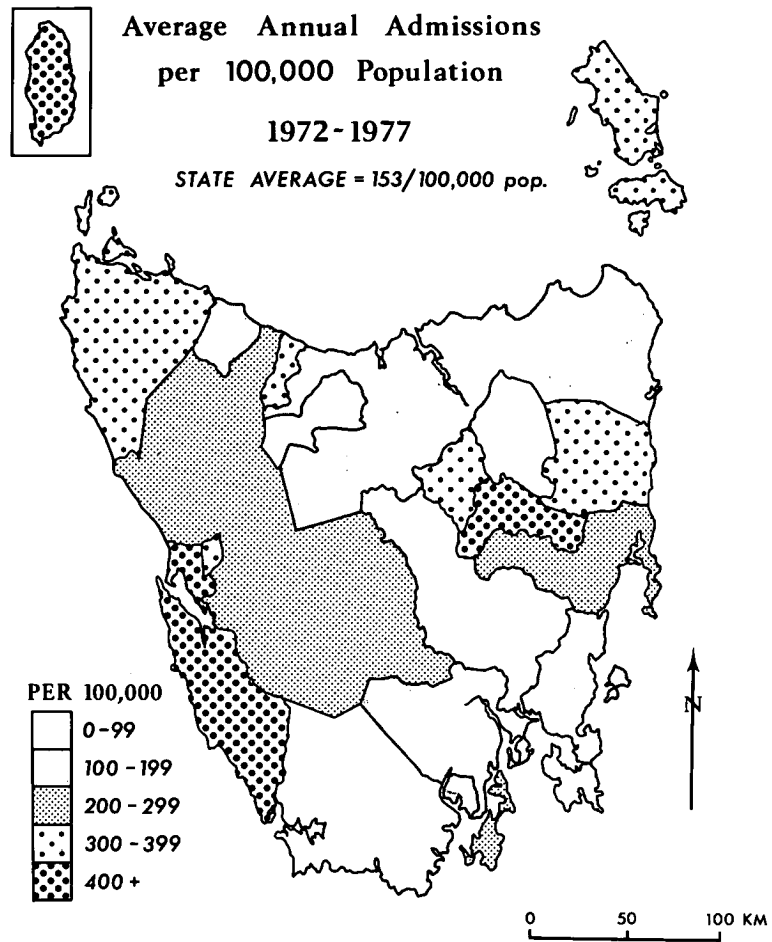


Figure 3.2.8 Hospital Admissions for Asthma in Tasmania 1972-1977 by Region.

could easily have been coded as Launcestonians. The patterns, although interesting, were difficult to interpret. Many factors had influenced them. Distance from a hospital and the availability of competent, sympathetic doctors were two major considerations. There was some evidence from the map that people living in isolated rural areas that contained hospitals used the hospital to a greater extent than those who lived in cities that offered a greater choice of health care alternatives. Confused with this pattern, however, were others. For example, a distance decay effect could be observed in non-hospitalised rural areas peripheral to city hospitals.

Regional Seasonality

When the average monthly admissions for the state or any of its sub-regions were graphed it was a simple matter to identify months of increased morbidity from those of lowered admissions. The state averages were included in the bottom left-hand corner of figure 3.2.9. The distribution was very flat and close to a non-seasonal monthly average, 8.7 per cent. March alone stood out with about 11.5 per cent of total admissions. Similarly to age-sex groups, it was hypothesised that different areas of the state, enjoying differing environments would exhibit different seasonal patterns. To examine this, local government areas were grouped into eight "natural regions" to ensure sufficiently large numbers for realistic monthly averages to be calculated. These were then used to construct histograms for mapping. The results are illustrated in figure 3.2.9.

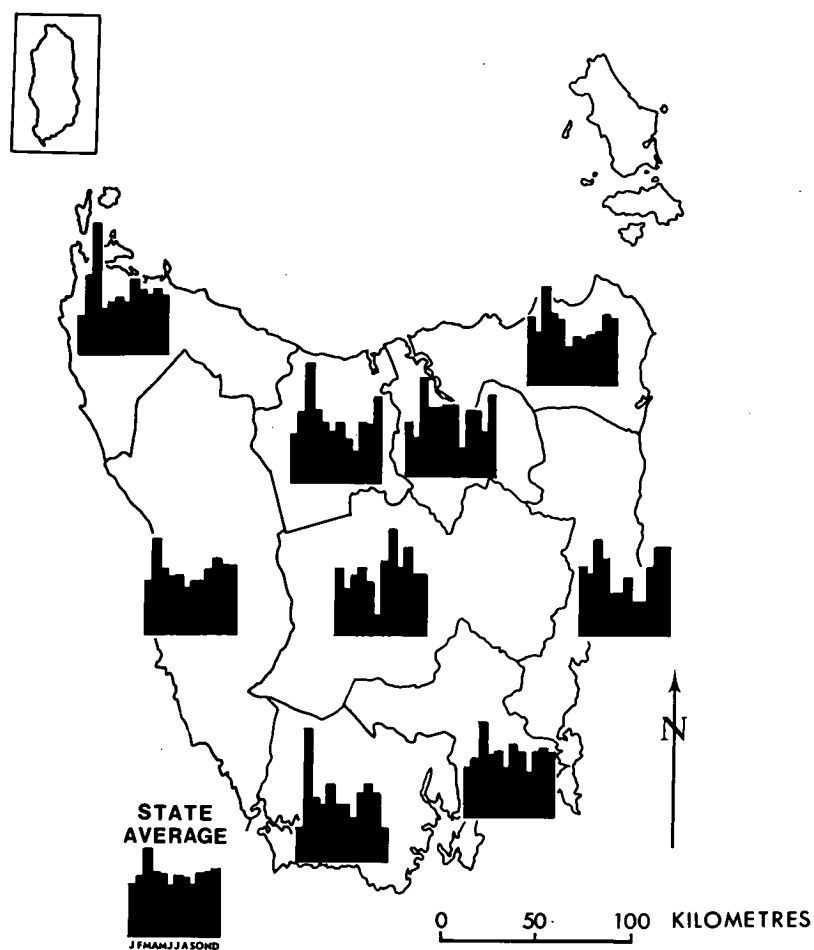


Figure 3.2.9 Average Monthly Admissions for Asthma in Tasmania 1972-1977 by Region.

It was immediately obvious that the state average was disguising a vast variation in local seasonality. Closest to the state average was the Hobart Metropolitan Area and its periphery. Its slightly higher March and July averages, +1 per cent, were the only differences. Five areas showed March peaks but in the West Coast and Channel areas this peak arrived a month earlier in February: in the Midlands it was missing altogether. The Northwest gave the strongest peak for March, seventeen per cent, followed by its neighbouring area Tamar, fourteen per cent. The Northeast had thirteen per cent of its admissions in March.

Winter admissions were generally average to low. On the East Coast the months of May to September were low. In the Midlands, June, was very low, 1.5 per cent, but rose to fourteen per cent in August. The Tamar region experienced a low in August, 3.5 per cent. In the Northeast, also, there was a low Winter value. Spring peaks were rare. The Midlands demonstrated one in October and the East Coast in October-November. The East Coast peak in Spring was accentuated by the Winter trough. Similarly, in the Channel area, lows in August and December highlighted the marginally above-average values in September to November. Early Summer peaks were evident in the Tamar, Northeast and East Coast areas. In contrast, the Channel and Northwest areas gave low values for January.

3.3 Hospital Admissions for Persons 0-9 Years of Age

Introduction

The most cursory glance at the material already presented on hospital admissions leaves an impression of vast inequalities across space, through time and between sexes and age groups. A concentration now, upon childhood asthma is not only indicated by the statistics of earlier pages but also reduces variation within the population with respect to occupational exposure, smoking, mobility and period of maximum residence, etc. It also brings this examination of hospital admissions into line with the other sections of the research programme that are concerned with asthma episodes in children.

Over the six years, 1578 hospital admissions were made for children in this age group. This represented forty-two per cent of all admissions for asthma. Over twice as many admissions were for boys as for girls; 1070 and 502 respectively. There were some annual variations in admissions and, although the sexes followed a similar pattern of high and low years, females demonstrated a much greater range (figure 3.3.1). In their monthly averages males showed the greatest variation, a large peak in March and troughs in June and January. Females did not demonstrate the same range but had peaks in March, October and December, and troughs in January June and September (figure 3.3.2).

Monthly averages, although informative, concealed the variation of any one month's admissions from year to year.

Figure 3.3.3 rectified this by giving monthly admissions for each

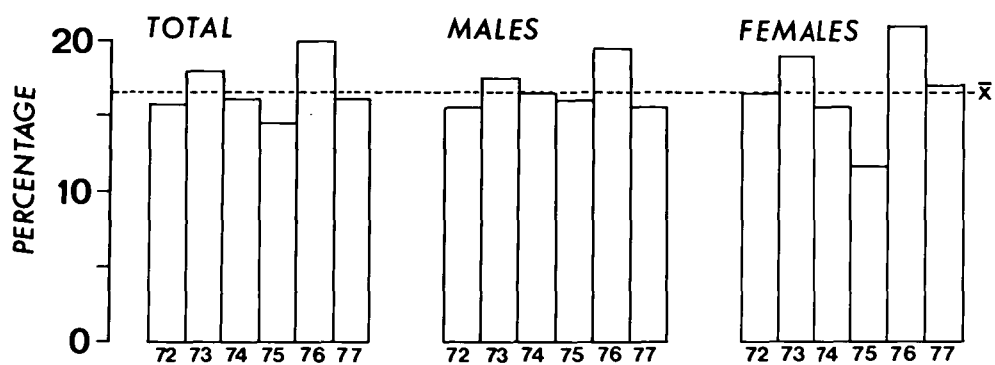


Figure 3.3.1 Percentage Distribution of Annual Admissions to Hospital in Tasmania 1972-1977 by Children aged 0-9 Years.

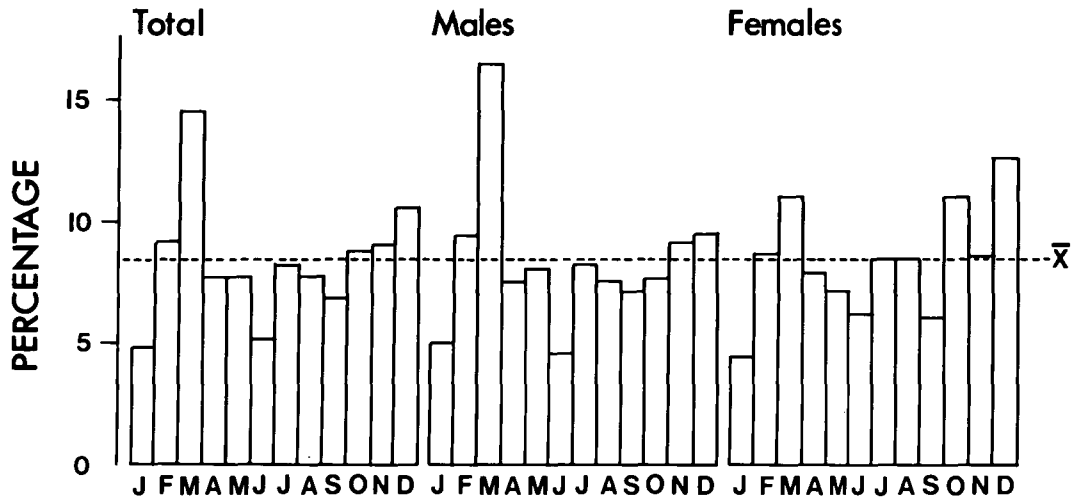


Figure 3.3.2 Average Monthly Hospital Admissions in Tasmania 1972-1977 for Children Aged 0-9 Years.

sex both as absolute numbers and as percentages of the total admissions for either sex in the six years of records. When 3.3.3.a and 3.3.3.b are compared, the dominance in absolute numbers of male admissions diminishes the variation in the female monthly series. Some concurrence is noted, for example peaks in December 1973, and March 1977. Opposite extremes were also obvious in March 1975 and in March 1976. The percentages of 3.3.3.c and 3.3.3.d made comparison much easier and revealed the relative variation in the female record to be as great as that in the male series. For portions of the series the two sexes appeared to be in phase and then were disturbed to return to phase later on. The major perturbation between the sexes seemed to be associated with the month of March. For example, in the eighteen months from January 1975 to June 1976, if the male admissions for March were reduced by seventy-five per cent the correlation between the time series would be much enhanced.

For 1977 only, admissions were categorised by day of the week. The percentage distribution is given in figure 3.3.4. The variation in children was greater than that for the entire population, (figure 3.2.3). Sundays had the highest proportion of admissions, 18.6 per cent, and Fridays the lowest, eleven per cent. Testing of the absolute frequencies against the expected daily frequency ($N=7$) gave a χ^2 value of 5.28 with six degrees of freedom. The "weekend" effect was therefore thought to be insignificant.

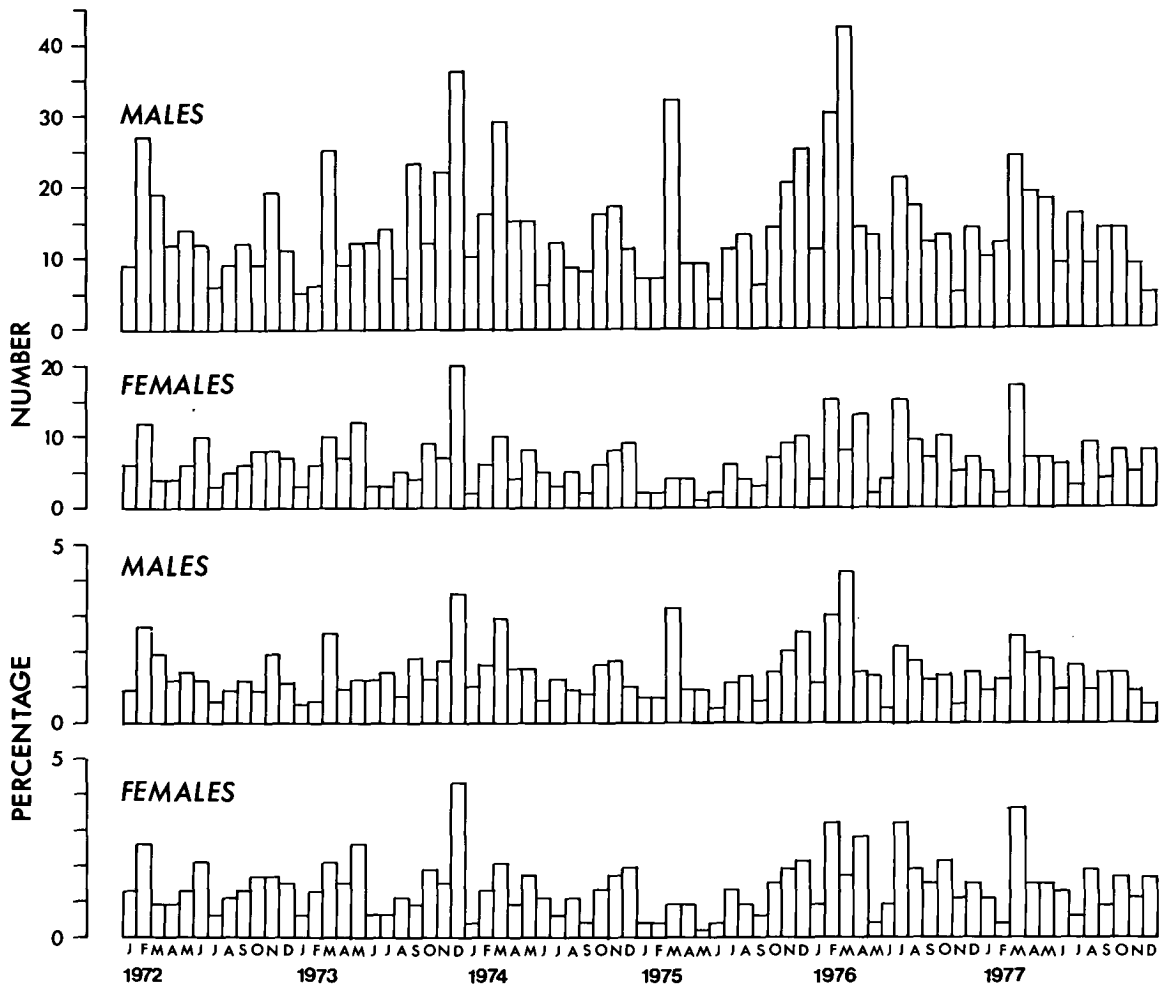


Figure 3.3.3 Absolute and Percentage Distributions of Monthly Hospital Admissions in Tasmania 1972-1977 by Children Aged 0-9 Years.

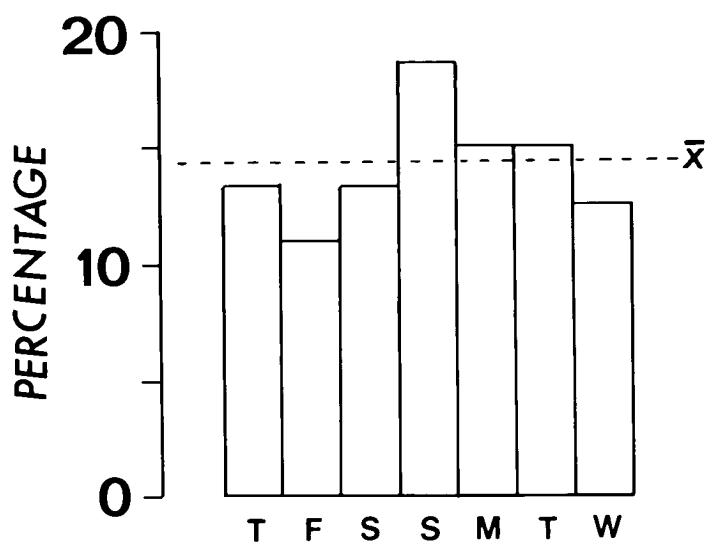


Figure 3.3.4 Percentage Distribution of Hospital Admissions for Asthma in Children Aged 0-9 Years by Day of Week.

Spatial Patterns

Using the local government area of residence, annual average age-specific admission rates were calculated for both sexes. Males had an overall rate of 467 admissions per annum and females had a rate of 227. These were based upon 100,000 persons of the same age and sex. Each local government area was mapped with its appropriate rate. To facilitate the comparison of maps using two different rates, location quotients were used. These used multiples of the norm in describing spread; the legends contained categories for twice and thrice the norm and a half and a third of the norm. Comparisons were made between areas upon how many times above or below average their admission rates fell (figures 3.35a & b).

When comparing maps of the two sexes one would expect that the social, economic and distance factors influencing the admission to hospital would be equal for either sex and that if these were the only important factors, that the maps would be fairly similar. Therefore, dissimilarities would point to differing aetiologies rather than different influences upon the decision process of hospitalisation.

Similarities between the two maps can be seen to reflect differential levels of access. The central suburbs of the Hobart Metropolitan Area are slightly above average in both maps, this quickly falls to below average in the peripheral L.G.A.s and to very low in some of the extremely isolated L.G.A.s. Asthmatics close to the hospital enjoy a slight over service and those further from hospital suffer from their distance. Generally, the

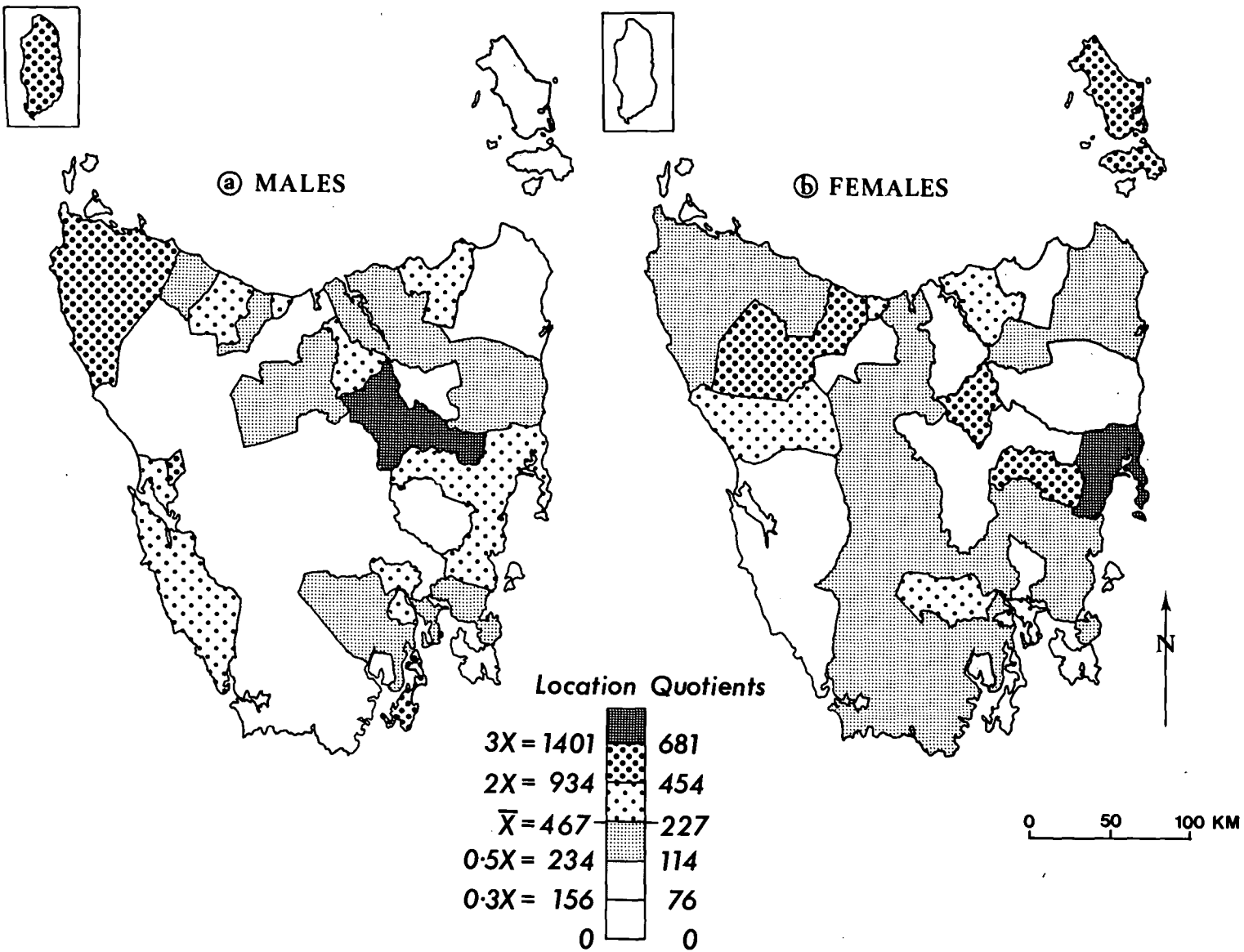


Figure 3.3.5 a & b The Regional Variation in Hospital Admissions for Asthma by Children Aged 0-9 Years in Tasmania 1972-1977.

patterns for Launceston, the Northeast, the North Midlands, the Midlands and the South are similarly low. Both sexes show a high admission area on the central East Coast that extends inland to a greater extent in the male map. One of the L.G.A.s Campbelltown, is puzzling; it is the highest category for males and the lowest for females. This major country town contains a hospital and access should not be a problem.

The major differences are in the Northwest and West Coast areas. Here male admissions are high and females low. A midsection that contains the West and lower Northwest Coasts is low for males and high for females. These revealed differences in morbidity between the sexes are inexplicable. Some factor(s) in the physical environment must affect the male and female childhood asthmatic differently. If this is the case, the seasonal patterns for each sex in the several areas should be markedly different. To investigate this hypothesis the monthly variation in the grouped L.G.A.s is mapped for each sex.

Because males in this age range had experienced over twice as many attacks as females, the numbers were much larger for males and therefore, reasonable monthly averages could be calculated for some of the less populous areas. This was not so for females, many of the areas given on the male map had insufficient numbers of female admissions and had to be combined with contiguous neighbours or ignored. The maps used the same L.G.A. groupings and were, therefore, comparable for most of the areas. The male map, 3.3.6, portrayed extra information for the

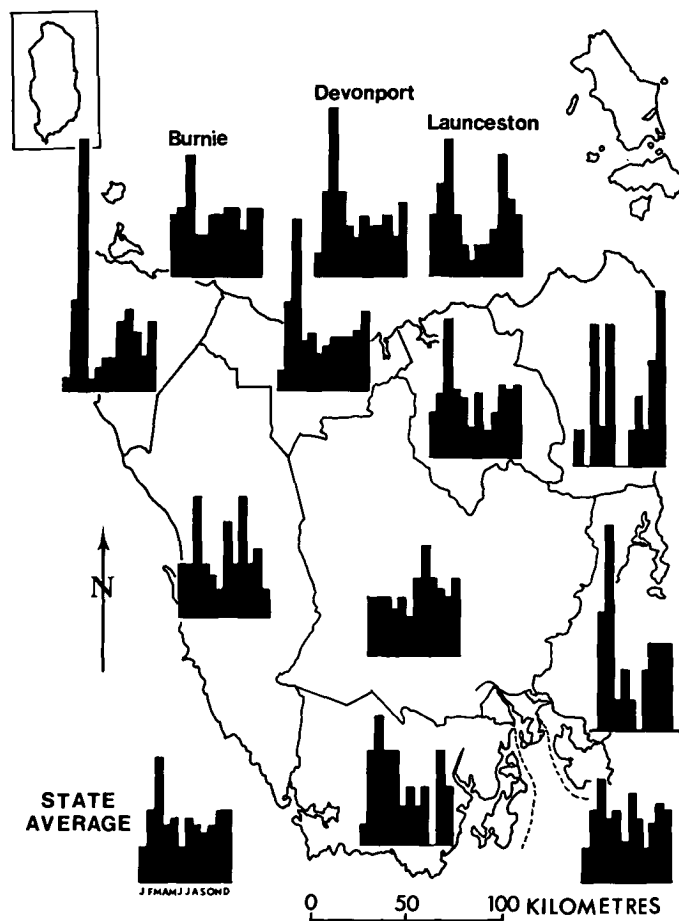


Figure 3.3.6 Average Monthly Admissions for Asthma in Tasmania 1972-1977 by Region for Males Aged 0-9 Years.

city of Launceston and the extreme Northwest and split the East Coast into northern and southern portions.

For males, every area exhibited some form of Autumn peak except the Midlands. This March peak generally exceeded all other peaks. It ranged from thirty-three per cent of all admissions in the far Northwest to slightly over thirteen per cent in the Hobart Metropolitan Area. In the Midlands, the admissions were about half of this low peak value, seven per cent. The second highest March peak was found in the southern half of the East Coast, twenty-seven per cent. The Northwest averaged twenty-two per cent with a range from eleven per cent (Burnie), to thirty-three per cent (extreme North-west).

March was the only month to stand out noticeably in the state averages. For some of the regions other months assumed importance. The northern East Coast had a Spring/Summer peak in November/December and Launceston a Spring peak in October. Winter peaks were found in three areas; the West Coast, twelve per cent, the Midlands, 14.5 per cent, and the Hobart area, twelve per cent. Urban areas tended to have much flatter monthly distributions than rural areas. Burnie was very close to the State average with its pronounced March peak. Devonport's March peak was even greater at the expense of other months. Launceston demonstrated an almost symmetrical distribution with peaks in March and October and a Winter trough. The Hobart Metropolitan area enjoyed the flattest distribution; its largest peak was March, thirteen per cent, followed by July, twelve per cent, and October, eleven per cent.

In general, female admissions (figure 3.3.7) showed much less range than their male counterparts. The Spring/early Summer peak was modal for the State and was also common in several areas. October accounted for twenty-five per cent of admissions in the far south and November, twenty-four per cent on the East Coast. The Midlands admissions peaked in August, seventeen per cent, and remained high in October, fourteen per cent. The flattest distribution was found, this time, on the Northwest Coast; April and October both peaked a maximum of eleven per cent of admissions. The two Northwestern cities, however, had Spring maxima. Burnie peaked in April and July, Devonport in March and December. Launceston's numbers were too low for good averages but the Tamar region showed a bimodal distribution with a maximum in December, 17.5 per cent, and a secondary in March, thirteen per cent. Hobart, again, was fairly flat. Its maximum peak was in December, fifteen per cent, followed by March, 12.5 per cent, and July, twelve per cent, and October, eleven per cent, a little peak in every season of the year.

On comparing the two maps, the differences between the sexes' seasonalities are marked. Male admissions were more strongly seasonal with peak admissions in the early Autumn. Female admissions had a more muted seasonal pattern in which Spring/early Summer peaks dominated. Both sexes showed geographic variation in their seasonality patterns. The most similar monthly distributions were for the Midlands; one of the flatter distributions with a peak in August. Interesting contrasts were

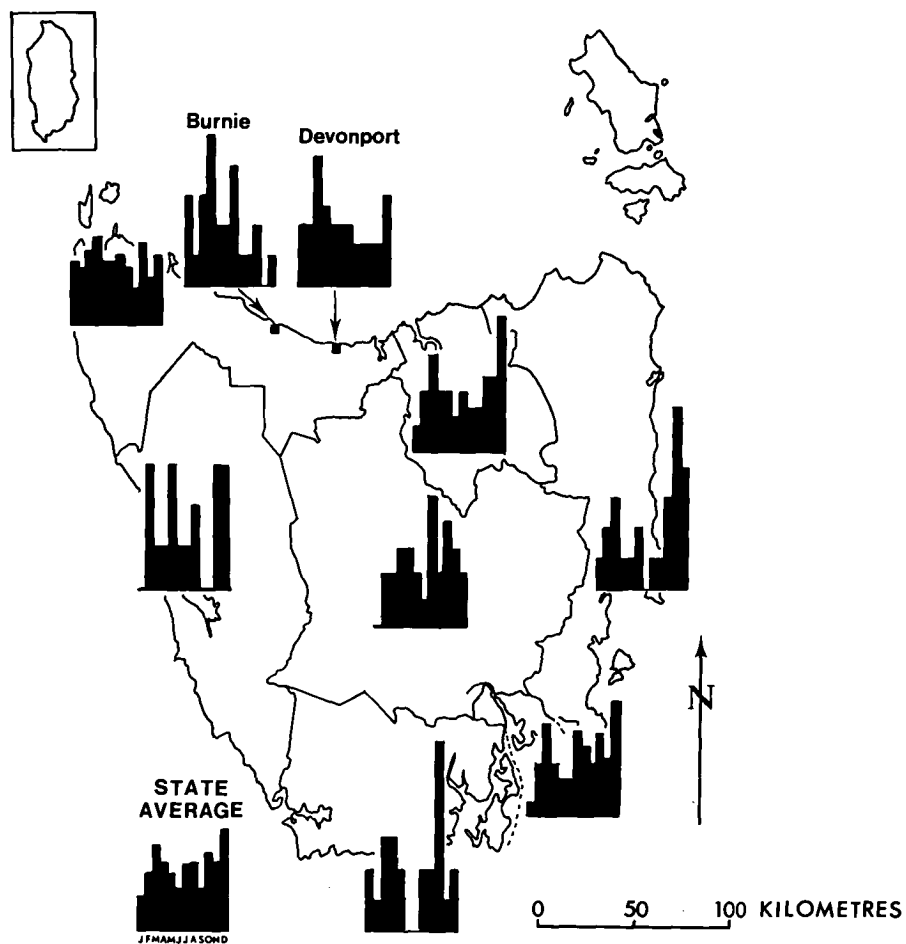


Figure 3.3.7 Average Monthly Admissions for Asthma in Tasmania 1972-1977 by Region for Females Aged 0-9 Years.

found in the average Northwest patterns, where a very flat female histogram contrasted with a strong Autumn peak in the male pattern. In both sexes, the Hobart area demonstrated a very low variation, on a monthly basis, with small peaks observed in any season. The patterns result from the multiple effects of factors such as climate, pollution, household heating and susceptibility to infections in addition to the various errors of data acquisition and processing which may have had considerable effects upon the monthly averages of the low population/low admission areas.

3.4 Analysis of the Relationships Between Hospital Morbidity and the Atmospheric Environment

Introduction

The many relationships that have been observed between morbidity from asthma and weather were noted in section one. Although repetitious, some of the literature is reviewed here in more detail with particular emphasis upon Australian works. All Australian studies of asthma biometeorology have used hospital admissions as their basic source of morbidity data. This section, therefore, has comparative value in that the processes illuminated by work in Perth or Brisbane can be usefully re-examined in light of Tasmanian experience.

The vast majority of work on Australian data has been that of E. H. Derrick and occasional collaborators. These seasonality studies, however, have been restricted to mainland capital cities particularly Brisbane. Here, hospital casualty attendances sometimes limited to night-time and/or children's attacks, were commonly related to data from a local meteorological station. The daily meteorological data were usually aggregated to weekly averages and correlated with weekly asthma attendances. Although some control over age was exercised by taking children's attacks, the sexes were never treated separately.

Seasonal patterns were examined by taking the weekly averages for principal cities and then "smoothing" the distribution by taking the three-week moving average.² This method showed a bi-modal Spring wave and a single large Autumn wave. Observed

in each of the cities, the waves were seen to be successively lagged from North to South with Melbourne a month behind Brisbane. Waves in February and/or March were also commonly observed. After considering several other possible causes the author concluded that the seasonal waves were probably due to a variety of interacting factors.

Some possibilities were examined by calculating co-efficients between the weekly means for weather data and asthma attendances. Derrick found that the weekly attendance rate was highly correlated, $p \leq 0.01$, with the mean temperature six weeks earlier;³ but that this did not represent a consistent effect. Often weekly means were exceptionally higher or lower than that expected from the lagged relationship with temperature. A close look at these exceptional weeks was taken by examining residuals from a regression of weekly means upon those six weeks earlier. High and low asthma weeks (greater and less than one standard deviation from the mean respectively) were analysed separately. Unfortunately, no one weather factor was found to be associated uniformly with either high or low asthma weeks. The most striking association was that of increased asthma with a cold dry change and low asthma in humid weather. Other factors were considered to be operating but were unknown.

In a later study⁴ Derrick analysed the twenty-six cooler weeks of the year separately. He discovered significant correlations between mean weekly asthma attendances and the mean weekly temperature, dew point and 3 p.m. relative humidity; with the greatest correlation at a five or six week lag. Other positive

correlations were found with rainfall and soil moisture (16 week lag) and a negative correlation with sunshine hours. In warmer weeks attacks significantly diminished inversely to the temperature, dewpoint, relative humidity, rainfall and soil moisture averaged over the current week. Cold dry changes between weekly averages were often related to an increase in asthma and in rainy periods asthma usually decreased.

In Perth, Western Australia, Hobday and Stewart⁵ used daily asthma attendances between April 1st and September 30th, 1968 for correlation analysis with weather parameters. In order to allow for a short lag in the effect of weather on asthmatics a three day lag was built into the data. Significant correlations were found between daily asthma attacks and pressure two days previously and ionisation three days before. The correlations found for daily data were much smaller (0.2-0.3) than those found for Derrick's weekly data. The significance levels were commensurately low.

In Sydney, Keig and McAlpine⁶ used thirteen months of daily data, June 1976-June 1977. They examined correlations for different averaging periods, ages and sexes. The asthmatics were divided into four groups; males/females and under fifteen/over fifteen. Each of these groups demonstrated significant relationships between their attacks and weather. Daily attendances during Spring were correlated with 3 p.m. wet bulb temperature, $p \leq 0.01$, at lag zero and minimum temperature and 9 p.m. wet bulb temperature with a one day lag. The significance of these correlations dropped to $p \leq 0.05$ with a lag of two days. No

significant correlations were found in the Autumn. Weekly attendances were correlated with weekly mean temperatures and the 9 a.m. and 3 p.m. wet bulb temperature. In Autumn and Spring the weekly attendance by children peaked one to two weeks earlier than the adults. Monthly attendances by children were significantly associated with 9 a.m. relative humidity ($p \leq 0.05$) for the current month. With a lag of one month, monthly adult attendance was positively correlated with minimum temperature and negatively correlated with the diurnal range of temperature for the previous month, $p \leq 0.05$.

These findings in the Australian literature are summarised in table 3.4.1. It is readily apparent that such relationships are influenced by many factors. There are of course, geographic differences. These are obscured by two effects. The first is the time segment used; series of the length of a year or greater yield different relationships to those of a season's length. Daily series for one season demonstrate different relationships to those from another season and so on. The second is due to the time averaging constant used; daily, weekly and monthly averages again portray different relationships. This effect can be modified considerably by additional "smoothing" techniques applied to the data, for example, moving averages applied to weekly means. It would seem to be an appropriate strategy for future research into hospital morbidity biometeorology to repeat analysis not only for different age/sex groups but also for different time averages, seasons of the year and extreme events. Applied to more than one geographic location, this approach should illuminate meteorotropic mechanisms more precisely.

Table 3.4.1
Australian Comparisons of Biometeorological Relationships with Asthma Morbidity

Weather Element	Correlation	Significance	Lag	Time Average	Location
Mean Temperature (WHY)*	-0.89	0.001	0	weekly	Brisbane
Mean Temperature (CHY)**	0.95	0.001	5-6wks.	weekly	Brisbane
9a.m. Dewpoint (WHY)	-0.8	0.001	0	weekly	Brisbane
9a.m. Dewpoint (CHY)	0.95	0.001	5wks.	weekly	Brisbane
3p.m. Relative Humidity (WHY)	-0.62	0.001	0	weekly	Brisbane
3p.m. Relative Humidity (CHY)	0.82	0.001	6-7wks.	weekly	Brisbane
Rainfall (WHY)	-0.75	0.001	0	weekly	Brisbane
Rainfall (CHY)	0.83	0.001	16wks.	weekly	Brisbane
Sunshine (WHY)	0.07	N.S.	0	weekly	Brisbane
Sunshine (CHY)	-0.74	0.001	0	weekly	Brisbane
Soil Moisture (WHY)	-0.58	0.01	0	weekly	Brisbane
Soil Moisture (CHY)	0.82	0.001	16wks.	weekly	Brisbane
Ionisation	-0.27	0.01	3 days	daily	Perth
Pressure	-0.21	0.05	2 days	daily	Perth
9a.m. Wetbulb	+	0.01	1 day	daily	Sydney
3p.m. Wetbulb	+	0.01	0	daily	Sydney
Minimum Temp.	+	0.01	1 day	daily	Sydney
Mean Temp.	+	0.05	0	weekly	Sydney
9a.m. Wetbulb	+	0.05	0	weekly	Sydney
3p.m. Wetbulb	+	0.05	0	weekly	Sydney
9a.m. Relative Humidity	+	0.01	0	monthly	Sydney
3p.m. Relative Humidity	+	0.05	0	monthly	Sydney
Minimum Temp.	+	0.05	1 month	monthly	Sydney
Diurnal Range	-	0.05	1 month	monthly	Sydney

* (WHY) Warm Half of the Year
 ** (CHY) Cold Half of the Year

Cross-Correlation Analysis of Tasmanian Data

Because of the low numbers involved in Tasmania when morbidity was measured on a daily basis, the distribution of daily admission values contained many zeros and was distinctly skewed. Five morbidity series were extracted from the data; the state total morbidity, male morbidity, female morbidity, child (under ten years) morbidity, Hobart area morbidity and Northwest area morbidity. They were cross-correlated with meteorological variables and indices using various lags.

First the correlations were calculated for synchronous relationships using zero lag. The analysis was then repeated for each pair of weather variable and morbidity series up to fifty lags. As fluctuations in asthma morbidity could not effect changes in the weather, only positive lags were used, that is, the weather variables were lagged on the stationary morbidity series. In this way the cross-correlation at a lag of fifty gave the relationship between the morbidity series and the weather fifty days previously.

An examination of a given series (see figure 3.3.3) emphasised the need to deseasonalise the meteorological data. This was accomplished prior to calculating the cross-correlation by forward differencing:

$$x_i = x_{i+1} - x_i$$

This, effectively, gave the daily change in the given meteorological variable. As well as removing seasonal fluctuations and trend, this exercise was valuable from a theoretical biometeorological point of view. It is thought to be the change in weather elements,

not their absolute value, that stimulates a meteorotropic response. Forward differencing supplied just those changes.

The cross-correlations for six years' of daily values proved to be insignificant. Occasionally, in one of the fifty lags a correlation of 0.1 would be found and because of the large N would be significant at $p \leq 0.001$. This level of correlation, however, was virtually meaningless. This was true for every permutation of morbidity index and meteorological parameter tested. The lack of correlation was thought to be due to perhaps four possible causes. First, it was thought that antagonistic relationships could exist between weather and asthma that varied within the course of a year and cancelled each other out. Second, it was possible that the physiological response to a meteorological stimulus might be triggered by durations of exposure greater than that of one day. Third, the daily numbers of morbidity episodes were few even for the state taken as a whole and it was believed that the preponderance of zeros and the non-normal distribution of scores could have contributed to the diminution of the coefficients. The fourth conclusion was that no relationship existed between daily admissions to Tasmanian public hospitals for acute asthma and the current or preceding fifty days of weather variations.

The evidence from the literature made the fourth possibility unacceptable. It was decided, however, to control for the other postulated effects by adopting three different methods of analysis; variation of time averaging, seasonal studies and an examination

of extreme events. The first technique, variation of time averaging, increased the stimulus-response period to accommodate any variation in reaction to weather stresses. The second method, seasonal studies, attempted to counteract any inter-seasonal antagonisms by calculating the weather-morbidity correlation matrix for different times of the year. Finally, extreme events were examined on the grounds that during peak events any environment-morbidity interaction would be at its highest level.

The Effect of Varying the Time Averaging

The low values of correlation obtained by using daily data were unchanged with up to fifty days lag between the weather and the morbidity data. Other research had demonstrated larger and more significant coefficients by using weekly and monthly averages. It was, therefore, suspected that any stimulus-response reaction in the Tasmanian data may have been based upon exposures to weather stresses greater than a day's length. This type of time relationship would not have been detected by lagging daily data.

To examine this problem more closely, different time-averaging constants were used to modify the daily values and the cross-correlations were re-calculated. The constants chosen were multiples of two except for the special case of seven days. In the first analysis, the daily meteorological series (x_i) were replaced by the two-daily averages (y_i) as follows:

$$y_i = (x_i + x_{i+1})/2, y_{i+1} = (x_{i+2} + x_{i+3})/2, \text{ etc.}$$

The morbidity series (m_i) were replaced by their two-daily series (n_i) as follows:

$$n_i = m_i + m_{i+1}, n_{i+1} = m_{i+2} + m_{i+3}, \text{ etc.}$$

Cross-correlation analysis was then performed and the time-averaging was then repeated for increments of four days, seven days, eight days, sixteen days and thirty-two days.

After each averaging procedure cross-correlations were calculated up to twenty lags for each weather-morbidity permutation. Correlations remained extremely low in each analysis. This low level was unaffected by forward-differencing. It was concluded that the time-average, and hence the exposure period, was not a contributing factor to the lack of correlation between the weather and morbidity series.

Seasonal Studies

Momiyama's study of seasonality of mortality demonstrated seasonal differences in the regression equation of the effect of temperature upon mortality.⁷ Her examples of variation in direction and degree of response within the course of a year suggested an explanation for the minute coefficients obtained from a series of six years' length. It was believed that relationships in one season were being cancelled by negative relationships in another. To investigate this further, the seasons were taken and analysed as groups of Springs, Summers, Autumns and Winters.

Tables 3.4.2a, b, c and d, illustrate the results of taking the seasons separately for correlation analysis. Eleven meteorological variables are averaged for the state by taking the values for Burnie, Campbelltown and Hobart. These are then correlated with morbidity indices for men, women, and children. In addition, Hobart morbidity is correlated with Hobart weather data

and Northwest morbidity is correlated with Burnie weather data. In the tables the meteorological variables are represented by state averages for the male, female, and juvenile morbidity groups and the site specific values for the geographical groups. It is noted that only one coefficient is greater than 0.2 and because of the large number of days involved this is significant at $p \leq 0.001$. The low correlations are, however, one order of magnitude greater than those found at the annual level.

In the Spring, male morbidity was related to wind. The daily wind run was negatively related to morbidity but the 3 p.m. windspeed had the opposite sign. Female morbidity stood out in this season and was primarily related to temperature. It was positively correlated with minimum, maximum, 9 a.m. wet-bulb, 3 p.m. dry-bulb, and 3 p.m. wet-bulb temperatures and was negatively related to 9 p.m. windspeed. Children's attacks were negatively related to minimum temperature, that is, the lower the minimum temperature the higher the level of morbidity. Hobart morbidity was related to the hours of sunshine and the Northwest's attacks were related to maximum temperature.

In Summer the weather relationships were fewer. Male episodes were related to afternoon temperatures, female episodes to morning windspeed and rainfall. Children's morbidity was related to high morning wet-bulb temperatures and to low afternoon dry-bulb and maximum temperatures and sunshine. Neither of the two geographic areas showed any significant correlation in this season.

The highest correlations were found in the Autumn. Men, women and children presented very similar pictures. Morbidity in

Table 3.4.2
Seasonal Correlations Between Weather and Hospital Morbidity

c) AUTUMN					
Weather Element	Males	Females	Children	Hobart	Northwest
Minimum Temperature	+0.13	+0.11	+0.18	+0.09	
9 a.m. Dry Bulb	+0.15	+0.11	+0.20	+0.09	
9 a.m. Wet Bulb	+0.15	+0.11	+0.19	+0.11	
9 a.m. Windspeed					-0.13
Maximum Temperature	+0.16	+0.09	+0.23	+0.09	
3 p.m. Dry Bulb			+0.15	+0.08	
3 p.m. Wet Bulb			+0.17	+0.09	
3 p.m. Windspeed		+0.11			
Sunshine	+0.15			+0.09	
Rain			-0.11		
Windrun					
d) WINTER					
Minimum Temperature		-0.14	-0.09		
9 a.m. Dry Bulb		-0.13			-0.08
9 a.m. Wet Bulb		-0.15			-0.08
9 a.m. Wind Speed		-0.11			
Maximum Temperature		-0.09			
3 p.m. Dry Bulb		-0.09			
3 p.m. Wet Bulb		-0.13			
3 p.m. Windspeed		-0.09			
Sunshine					
Rain		-0.07	-0.10		
Windrun					
a) SPRING					
Weather Element	Males	Females	Children	Hobart	Northwest
Minimum Temperature		+0.17	-0.13		
9 a.m. Dry Bulb					
9 a.m. Wet Bulb		+0.15			
9 a.m. Windspeed		-0.14			
Maximum Temperature		+0.13			+0.17
3 p.m. Dry Bulb		+0.14			
3 p.m. Wet Bulb		+0.12			
3 p.m. Windspeed	+0.13				
Sunshine				+0.13	
Rain					
Windrun					
b) SUMMER					
Minimum Temperature					
9 a.m. Dry Bulb					
9 a.m. Wet Bulb			+0.10		
9 a.m. Wind Speed		+0.11			
Maximum Temperature			-0.12		
3 p.m. Dry Bulb	+0.10		-0.10		
3 p.m. Wet Bulb	+0.10				
3 p.m. Windspeed					
Sunshine			-0.14		
Rain		+0.10			
Windrun					

Correlations with $p > 0.05$ are not given.

these three groups and in the Hobart area was related to both morning and afternoon temperatures and the extremes of temperature especially the daily maxima. Other related variables were high levels of sunshine and low levels of rain. The highest coefficient was found to be between juveniles' morbidity and the maximum temperature, $r = 0.225$, $p \leq 0.001$. For the Northwest, taken as a whole, the only relationship shown was a negative one to 9 a.m. windspeed.

The correlations dropped in degree and number during the Winter months and all that there were found were negative. No correlations were described for the male or Hobart morbidity series. The Northwest morbidity was related to low morning temperatures. Children's episodes were related to lack of rain and low minimum temperatures. The female morbidity, however, demonstrated many correlations. These were with low temperatures, low windspeeds and low rainfall.

Winter and Spring were dominated by the correlations with female morbidity. These were negative in Winter and then positive in Spring. Summertime did not give a strong picture for any group and Autumn, conversely, demonstrated strong patterns for almost every morbidity aggregation that was considered.

To discover if weather-morbidity relationships were of a shorter duration than the three months used for the seasonal correlations each month was analysed separately. Taking the six months of January together, for example, there were 186 days of measurements upon which to base calculations. The correlations found at this level were only marginally higher than those found

for seasonal periods. Two months, April and October, the mid points of Autumn and Spring respectively, demonstrated no significant correlations between weather and morbidity.

Significant findings were summarised in table 3.4.3.

From late Spring to late Summer (November-February), the correlations with temperature and sunshine variables were negative. That is, high temperatures were correlated with low morbidity and vice versa. In November this was related to female morbidity; thereafter childhood morbidity dominated. Throughout the Autumn the correlations were with sunshine hours, rainfall and 3 p.m. windspeed. All of these relationships were positive. High values for sunshine were correlated with high values of male, Northwest, or Hobart area morbidity. High afternoon windspeed and rainfall was associated with high female morbidity. Early Winter was dominated by correlations with female morbidity, late Winter by those with childhood morbidity. Here, as in Summer, the relationships were negative. In September, at the beginning of Spring, the correlations were, again, positive. Interestingly, only the Hobart morbidity gained significant correlation at this time.

So in the course of a year, and even within the course of one season, the weather-morbidity relationships were seen to change for the groups' morbidity series separately and to change both in kind and degree. The monthly correlations were based on six years' accumulation of each single month. It was possible that the adding together of months from different years could have produced counteractive effects similar to those discovered when

TABLE 3.4.3

Monthly Correlations Between Weather and Hospital Morbidity

<u>MONTH</u>	<u>WEATHER VARIABLE/MORBID POPULATION/CORRELATION*</u>
January	9am Windspeed/Children/0.22 3pm Wet Bulb/Children/0.19
February	Maximum Temperature/Children/-0.21
March	3pm Windspeed/Females/0.20 Sunshine/Males/0.29 Sunshine/Northwest/0.23 Rain/Females/0.2
April	
May	Sunshine/Hobart/0.19 Sunshine/Northwest/0.20
June	9am Dry Bulb/Females/-0.23 9am Wet Bulb/Females/-0.18 3pm Dry Bulb/Females/-0.20 Minimum Temperature/Females/-0.25
July	3pm Wet Bulb/Children/0.19 9am Windspeed/Hobart/-0.24
August	9am Dry Bulb/Children/-0.20 9am Wet Bulb/Children/-0.18 3pm Dry Bulb/Children/-0.20 3pm Wet Bulb/Females/-0.19 Minimum Temperature/Males/-0.18 Minimum Temperature/Children/-0.17 Maximum Temperature/Children/-0.21
September	9am Dry Bulb/Hobart/0.18 9am Wet Bulb/Hobart 0.18 9am Windspeed/Hobart/0.18
October	
November	Sunshine/Females/-0.18 9am Wet Bulb/Females/-0.19
December	Maximum Temperature/Children/-0.23 3pm Wet Bulb/Children/-0.19 Sunshine/Females/-0.20

*Correlations with $p > 0.01$ not included.

series longer than a season's length were analysed. The lack of correlations in April and October were particularly disturbing in this regard. In some years these months may have demonstrated positive environment-weather relationships and in other years the opposite. Taken together these would have cancelled each other to give near-zero correlations. To investigate this problem further, the original graph of admissions by month and year (figure 3.3.3) was examined and "peak" months were selected for scrutiny.

Extreme Events

Figure 3.3.3 demonstrated the enormous variability in morbidity from month to month across the six year time span. Boys peaked every March and shared peaks in February 1972, March 1977 and December 1973 with the girls. Girls, in addition, peaked in April 1973, February 1976, April 1976 and July 1976. These peaks were defined as being greater than one standard deviation above the mean. Troughs below one standard deviation beneath the mean usually occurred during June or December for boys and during January or May for girls. The most prominent events occurred during December 1973 and February-March 1976. These occasions warranted closer examination.

The daily data for these months were reviewed and heightened morbidity was detected in both of the short series. The limits of the clusters were defined by the closest days before and after the event that possessed zero morbidity values. For December 1973, this involved the entire month of thirty-one days.

In February-March 1976 there seemed to be two peaks so correlation analysis was run upon the two together, thirty days, and the first one alone, twenty days. Cross-correlations were calculated for males, females, children and the total Hobart morbidity series, with respective meteorological series for Hobart.

The results are tabulated in table 3.4.4. Differences between men and women and adults and children are striking. During the December episode the correlations are reversed from those found during the March event. In December, there are only weak correlations with male morbidity; female morbidity, however, seems to be related to low maximum temperature, high relative humidity at 3 p.m. and low amounts of sunshine. The additional signs for increased minimum temperature and rainfall would tend to associate female morbidity with frontal weather conditions. Childhood morbidity follows the same pattern as the females' with only slight differences in size and degree. For Hobart generally, morbidity is related to the high maximum temperature and low wind speeds associated with anticyclonic inversions.

In February/March 1973 the situation changes. Male morbidity is related to maximum temperature, minimum temperature and 3 p.m. relative humidity suggesting that the effect may be due to thermal stress. Female morbidity is related, on the other hand, to the increased minimum temperature and 9 a.m. relative humidity connected with periods of rainfall. Childhood morbidity correlations for this time period do not match the females'. It is negatively related to rain and windspeed and positively related to temperature and humidity. Hobart morbidity, particularly when

TABLE 3.4.4

Correlations Between Hobart's Weather and Hospital Morbidity Series in December 1973, February-March 1976 and February 1976

Population: Time Period:	MALES			FEMALES			CHILDREN			HOBART		
	a	b	c	a	b	c	a	b	c	a	b	c
<u>Weather Element</u>												
Maximum Temperature		0.25	0.35	-0.20			-0.36	0.29	0.55	0.22	0.19	0.31
Minimum Temperature		0.26	0.24			0.31		0.43	0.51			
9am Relative Humidity					0.29			0.33	0.36			
3pm Relative Humidity			0.25	0.34			0.29	0.26	0.28			0.23
Sunshine				-0.42			-0.32					
Wind Run						0.35			-0.40	-0.32		
Rain					0.31	0.33	0.25	-0.20	-0.41			-0.21

Time Period: a = December 1973 (30 days)
 b = February-March 1976 (30 days)
 c = February 1976 (part) (20 days)

Coefficients (r) < 0.1 are excluded

If $N = 30$, $p = 0.05$ when $r \geq 0.35$, $p = 0.01$ when $r \geq 0.45$.

If $N = 20$, $p = 0.05$ when $r \geq 0.42$, $p = 0.01$ when $r \geq 0.54$.

the shorter, maximal peak correlations are examined, is related to high maximum temperature, low 3 p.m. relative humidity, high evaporation and low rainfall.

The contrasts in coefficients between the extended and shortened series for February-March 1976 are always due to a change in degree not the sign of the relationships. This underlines the finite and time-specific nature of any meteorotropic relationship in the data. The associations last for a short time and values to either end of the time period can dilute the coefficients if they are included in the analysis. Whatever relationship exists between a given weather element or synopsis and a morbidity series enjoys a peak period that quickly diminishes. The daily meteorological data for the two peaks are therefore re-examined and compared with daily synoptic weather charts in an attempt at pattern identification during the two peak periods.

Example 1

In February 1976, the peak event started on the twenty-second and high levels were maintained until the twenty-second of March. On the twenty-second of February an anti-cyclone that had been stationary over Tasmania for two days moved to the East over the Tasman sea and allowed the incursion of warm, moist, equatorial air from the Australian East Coast. The unusual blocking conditions produced by this air mass persisted for approximately three days, (figure 3.4.1). During this time sunshine hours dropped from 12.3 to zero, diurnal temperature range dropped from almost ten degrees to three-and-one-half degrees and humidity rose from a 9 a.m.

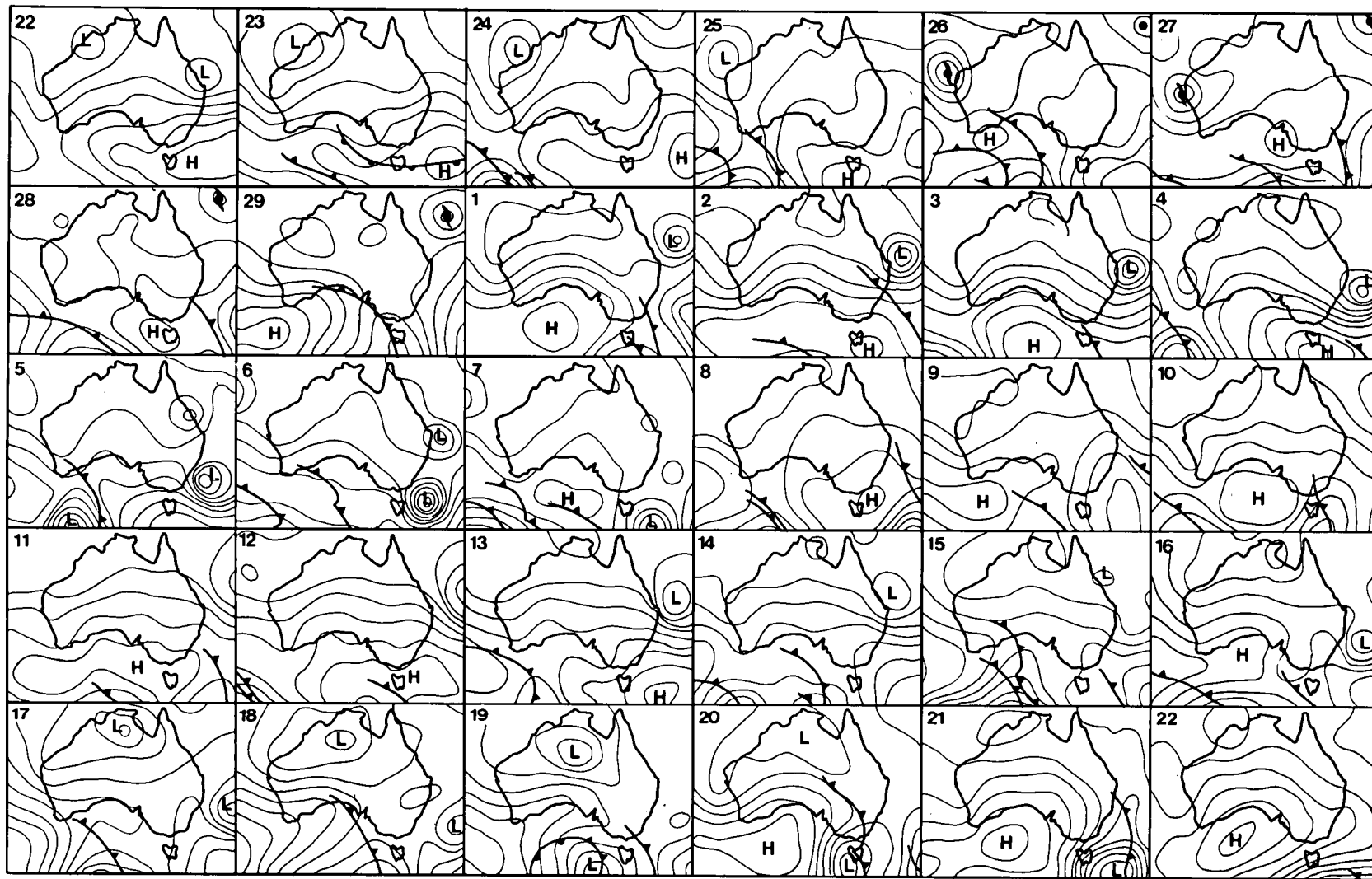


Figure 3.4.1 The Synoptic Progression During February-March 1976.

average of sixty-nine per cent to ninety-four per cent and an afternoon average of fifty-nine per cent to eighty-three per cent. This situation was alleviated by the twenty-sixth of February when two cold fronts in quick succession brought about rapid change. By the twenty-seventh another anti-cyclone was stationed over Tasmania bringing about sunshine and high temperatures. This was quickly changed on the twenty-ninth by a strong cold front from the West. The month of March was characterised by the southerly movements of decaying equatorial cyclones. Cyclone "Colin" caused widespread cloud cover, rain, high humidities and a low temperature range for five days from the third to the seventh of March. From the eighth of March until the thirteenth an anti-cyclonic pattern persisted and then moved East onto the Tasman Sea and allowed cyclone "George" to advect warm, humid air southwards. From the fourteenth to the nineteenth of March a westerly pattern of anti-cyclones separated by small cold fronts was established. This was broken on the twentieth by a southerly cyclone moving northwards over Tasmania bringing lowered temperatures and rain. This in turn was followed by clear anti-cyclonic conditions with a high pressure centre established over the Great Australian Bight.

Example 2

The month of December 1973, the other selected peak period, started with a cyclone stationed over the Bight, (figure 3.4.2). This in conjunction with an anti-cyclone off the East Coast of Australia, produced a strong southerly advection of hot continental air across the Bass Strait to Tasmania. On the second of December,

maximum temperatures peaked at 30.8 degrees in Hobart. By the third the cyclone had moved to stand over Tasmania, the maximum temperature had dropped twelve degrees and the average wind speed had increased from seven to twenty-five kilometres per hour. This major weather event was followed by several days of westerly anti-cyclones separated by single cold fronts. An anti-cyclone then established itself over the Tasman Sea and humid, Easterly winds with associated cloud dominated the State's weather. An approaching cyclone over the Australian Bight made the winds back to the North and Continental air was once again advected over Tasmania. The maximum temperature rose to 32.5 degrees, the 3 p.m. relative humidity dropped to thirty-eight per cent and the sun shone for 13.3 hours on the fourteenth. The next day, the fifteenth, the cyclone passed over Tasmania, the maximum temperature dropped thirteen degrees, the 3 p.m. relative humidity rose to one-hundred per cent and only half-an-hour of sunshine penetrated the cloud cover. The diurnal range in temperature changed from over seventeen degrees to just over two degrees. These conditions persisted for four to five days and then altered when a strong cyclone invaded the state from the West on the twenty-first and twenty-second bringing rain. This was followed by a westerly anti-cyclone pattern that was broken at the end of the month by a stormy cold front.

This examination of the synoptic processes involved during the two peak periods helps to elucidate the role of weather patterns in stimulating acute attacks of asthma. It underlines the importance of strongly fluctuating weather elements by relating these to

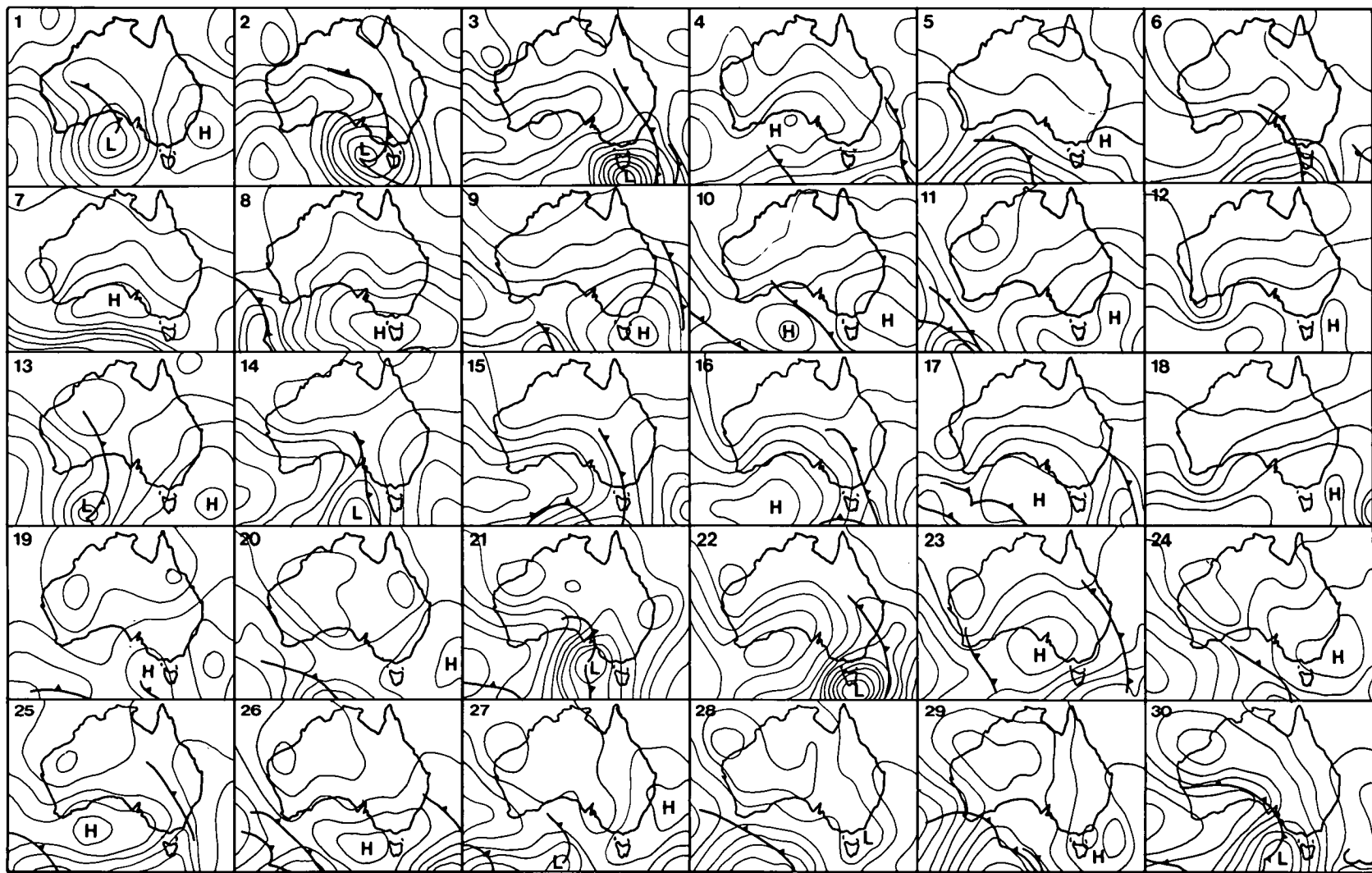


Figure 3.4.2 The Synoptic Progression During December 1973.

macro-scale atmospheric conditions. The nature of these relationships emphasises the value of Tasmania as a biometeorological laboratory. Peaks of asthma attacks occur most often when the regular flow of anti-cyclones delineated usually by single cold fronts is interrupted by a major influx of air either from the Australian continent or the Southern ocean. The sudden and gross change produced by the appearance and removal of these frontal air masses seems to be the culpable irritant that provokes epidemics of acute attacks. These synoptic situations arise more frequently and strongly in Spring and Autumn; Summer and Winter are usually dominated by westerly systems. The strong fluctuations and North-South air mass movements may be responsible, therefore, for the seasonal fluctuations in morbidity in Tasmania.

3.5 Summary

This section's examination of hospital morbidity data has led to the rejection of two of the null hypotheses stated in section one: that no significant temporal variation existed in any subset of wheezers, and no significant time relationship existed between any morbidity series and any physical element's series. First, significant temporal variation has been demonstrated in the time series of hospital admissions due to asthma episodes. Asthma morbidity, as measured by these acute attacks, has demonstrated a distinct seasonal pattern with peaks in Autumn and Spring. These seasonal fluctuations have been seen to vary for the different sexes and age strata. Childhood attacks have dominated the hospital admissions and have been examined separately. Boys have had twice as many episodes as girls and these have been concentrated in the Autumn, whereas, girls' attacks have been more pronounced in the Spring. Urban dwelling asthmatics' monthly distributions have tended to be deseasonalised. Rural-coastal dwellers have given the strongest seasonal fluctuations. Inland asthmatics were the only group to have exhibited a Winter peak in an otherwise flattened distribution.

Second, the different temporal and spatial patterns given by the sexes have been reinforced by an examination of weather-morbidity interactions. Significant correlations have been discovered between weather elements and morbidity. Correlations have been found, however, only when the time period used for their calculation was of a season's length or less. Both the direction and degree of correlation were discovered to change from month to

month and to differ between population sub-groups that had been based on characteristics of sex, age or residence. An examination of two peak months in the six years of morbidity has emphasised the role played by strong weather changes. These changes were seen to be characteristic of the Autumn and Spring seasons in Tasmania and usually involved major latitudinal transfer of air masses. During these times the male and female responses were seen to differ. Children's morbidity, dominated by high numbers of boys, closely resembled the male pattern.

These findings both corroborated and complemented the studies of asthma morbidity and weather that had been conducted in mainland capital cities. The low magnitudes of the coefficients were probably due to the lower total numbers of asthmatics in Tasmania compared to the mainland cities. This was a function of population size rather than relative prevalence. The low numbers, unfortunately, precluded any but the most crude population subdivisions for cross-correlations on a daily basis. For example, regional series could only be compared at the expense of any age-sex segregation and vice versa. The existence of significant correlation between weather elements and asthma morbidity, however, emphasises the importance of more detailed study of such effects. If the inaccuracies, limitations and unknowable biases of hospital morbidity statistics are only guessed at, the occurrence of any significant correlation must be viewed as extraordinary.

Controlled, prospective experiments are the only way in which the suggestive correlations stemming from these retrospective and poorly standardised studies can be tested. The elegance of

bimeteorological field experiments can be enhanced by using standardised methods to measure the morbidity fluctuations in homogeneous population samples from different geographic locations or demographic strata. These experiments yield data designed to test specific hypotheses. The next section describes one such experiment, selectively conducted upon samples of asthmatics of the same age, residing in different parts of Tasmania.

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SECTION 4

A BIOMETEOROLOGICAL CASE STUDY

4.1 Introduction

A considerable body of evidence has been presented from the literature, from the Tasmanian Asthma Survey and from hospital morbidity statistics, in support of environmental interaction with asthma morbidity. Significant spatial and temporal variations in morbid episodicity have been demonstrated. In most cases the data only permitted retrospective dissection. They had not been collected with either geographic or biometeorological analysis in mind. In addition much criticism could be levelled at attempts to use hospital morbidity statistics to detect spatial differences in temporal patterns. The spatio-temporal inequities in these data have been discussed in section three. Nevertheless, the value of the analyses to date has been in their exploratory nature and first approximation level of explanation. The associations and patterns inherent in the various data matrices have taken the overall schema one step further, from questions of: "Where?", and "When?", to questions of: "Why there?", and "Why then?".

Having established the gross patterns of association in space and time between elements of the physical environment and indices of morbidity, there remained the task of describing these

adduced relationships more perfectly. An experiment was therefore devised to test the validity of meteorotropic response relationships in asthma morbidity in Tasmania. This short-term, prospective, biometeorological study of asthma morbidity in similar populations at risk, living in dissimilar environments within the state, was structured to build upon the geographic analyses from earlier sections. Such structure, however, was necessarily modified by the limitations imposed by variations in population distribution, accessibility and the co-operation of asthmatics living in the study area.

After a review of biometeorological methods, that places this section in its theoretical and methodological context, the design of the case study is discussed in detail. This discussion includes the selection of study areas, the choice of asthmatic samples and the collection of morbidity and meteorology data. Next the asthmatic samples are compared on the basis of their health history, spirometry and residence. Following sub-sections describe various analyses of the relationships between morbidity and the physico-chemical nature of the atmosphere arising from the prospective records specifically compiled for the purpose during the study period. Finally, the findings for each area are summarised and discussed.

4.2 Biometeorology - A Review of Methods and Objectives

Biometeorology is a broad sub-field of investigation embracing by the disciplines of medical geography, meteorology, physiology, the life sciences, psychology, epidemiology and many other academic faculties and professions. A definitive work "Medical Biometeorology" was given by Tromp in 1963.¹ He, in turn, acknowledged the influence and work of Petersen² and De Rudder³. Their formative influence has continued to promote interdisciplinary research by workers in disparate specialities. Licht, a physician, produced "Medical Climatology"⁴ in 1964. Lowrey, a meteorologist, contributed "Weather and Life" in 1968. Munn, another meteorologist, published "Biometeorological Methods"⁶ in 1970 and Landsberg's "Assessment of Human Bioclimate"⁷ appeared in 1972. It was not until 1974 that Hunter, a medical geographer, pointed out the lack of contributions to this field from members of his own discipline.⁸ This lack was seen to be particularly surprising as medical geography appeared to be both well-suited and well-equipped for the exploration of climate and weather effects upon health and disease.

Biometeorology's modern foundations were built at the 1956 Symposium of the International Society of Biometeorology.

At this inaugural meeting the field was defined thus:

"Biometeorology comprises the study of the direct and indirect interrelations between the geophysical and geochemical environment of the atmosphere and living organisms, plants, animals and man..."⁹

Tromp took this definition and proceeded to subdivide and to classify the discipline according to subject and method.¹⁰ Five divisions were delineated: phytological, zoological, human, cosmic and palaeo-biometeorology. Human biometeorology, the subfield of primary importance to this work, was further divided into four major components; physiological, pathological, social and urban. Physiological biometeorology describes the influence of weather and climate on physiological processes in healthy individuals. Pathological biometeorology measures the influence of weather and climate on physiological and pathological processes in diseased individuals. Social biometeorology studies the effect of weather and climate on the social habits of man. Urban biometeorology investigates the effects of microclimates found in environments created by man; his buildings, towns and cities.

Methodologically, biometeorology falls into two major parts: empirical and experimental. Empirical methods emphasise the collection of data on physiological and pathological phenomena related to or suspected to be related to climate or meteorological factors without any preconceived hypothesis. They are concerned with forming hypotheses. Experimental methods are generally designed to test hypotheses and usually try to control for as many factors as possible. The two methodologies are complementary - many experimental designs are based on hypotheses formulated from empirical induction. Tromp also classifies biometeorological research by the type of approach that is adopted: indirect or direct. The indirect, or medical geographic approach involves the

mapping of geographical distributions of morbidity and a comparison of their spatial distributions with those of climatic averages. Depending on the community prevalence of disease mortality/morbidity this approach can then be elaborated by replicating the method for different age-sex strata of the population. The direct or biometeorological approach analyses two synchronous data sets, meteorological and clinical, in order to describe more exactly the relationships between them.

In an explanation of the slow scientific progress in the discipline, Tromp highlights two important problems. The first concerns the variability in "morbidity limit"¹¹ between individuals and in the same individual over time. This concept of a variable threshold that has to be passed before morbidity is perceived and acknowledged emphasises the need for surveillance of large numbers of cases to cancel the effects of individual resistance. For example, an individual who has experienced an episode of morbidity only recently may have a higher threshold, a temporary immunity, to a quickly repeated environmental insult; but in a continually monitored, large group of sensitives the average or proportional morbidity response will remain fairly stable. In a heterogeneous syndrome, like wheezy breathing, individual variation in meteorotropic¹² response may be very high and a statistical correlation based on large numbers is a better approximation of the relationship. On the whole, it is considered that if large, homogeneous, population samples are used for the clinical data one can expect to obtain reasonably-accurate measurements of morbidity fluctuations. Then, when the interrelationships are

established empirically, experimental research can proceed at the level of the individual.

Having established that the best measure of morbidity is the proportion of a constant, large group of sufferers that experience symptoms in any synchronous time segment, Tromp's second problem of method centres on the temporal lag, if any, that is to be used in the correlation of morbidity with meteorological measurements. Different meteorological elements are shown to have a lag in their clinical effect producing various correlations of the order of weeks before the onset of morbidity.¹³ A few single meteorological elements and their imputed relationships with asthma morbidity were described in section one. Actually, co-variance of asthma morbidity and any one meteorological/atmospheric element is likely to have low explanatory power. Each geophysical and geochemical parameter of the atmosphere is intimately related to every other. And, furthermore, the strengths and directions of these relationships may vary enormously with geographical location, and over time. Some elements may effect morbidity additively, others may act synergistically and yet others may be antagonistic in effect. Another question that has to be resolved is at what point in the morbidity episode are the measurements for correlation to be made; the beginning, maximum or conclusion? The myriad sources of error and bias both in morbidity data collection and in their analysis with meteorological elements call for extreme caution and careful planning. Biometeorological research, in addition to these substantive problems, is also plagued by insufficient

co-operation between disciplines and professions; lack of statistical expertise; a paucity of good biostatistical data and the popular abuse of climatic averages.

An ideal biometeorological field experiment would be based on geographical areas highlighted by the "medical-geographic" approach. This indirect analysis would be the initial stage of a research programme. The second stage would include the selection of homogeneous samples of "sensitives" in the several geographic locations. Tertiary activities would involve the synchronous measurement of suspected stimuli (weather, pollution, etc.) and the morbidity response (in this case wheezing). The fourth phase would manipulate the data set along various analytical pathways to sort out the various effects in the interaction matrix. Finally, given the relationships between morbidity and meteorology in different locations, it would become possible to predict morbidity variations from the appropriate environmental variations. The efficiency of such predictive models could be evaluated by a comparison of the predicted series with a monitored series of morbidity levels.

Such first-stage medical geographic analyses have been conducted in Tasmania and are recorded in section two. Spatial patterns illustrated there give strong indications of the potential of study areas. The selection of areas for biometeorological and ecological study based on their environmental contrast is well supported by other workers.¹⁴ It is commonly accepted that valuable insights can accrue from an examination of extremes in either the domain of space or the domain of time.

In regard to the choice of homogeneous population samples in which to measure morbidity fluctuations, the use of children is highly recommended.¹⁵ By monitoring asthmatic children living in special schools, Tromp¹⁶ selected an excellent biometeorological data source. Their use, in general, is justified on the basis of their increased homogeneity, their increased likelihood of maximum residence in one location and their increased sensitivity to environmental stress compared to that of adults. Occupational exposure and some of the manifestations of social class are assumed to be of minor importance with this age group. This would be particularly true of children in a residential school.

Having chosen sample populations based on some geographic rationale, the next task is to collect the two sets of data, meteorological and clinical. Meteorological data is usually obtained from a nearby weather station. This is adequate for most purposes particularly if the station is close to the population being monitored. Often, however, grades of weather stations exist and data may be available only for a limited number of measurements and for limited hours and days. Some parameters of the physico-chemical nature of the atmosphere are not measured routinely. The researcher interested in pollution and ionisation, for example, often has to measure such elements himself. The problems of instrumentation and recording, the choice of sampling periods and the selection of sampling sites are not trivial matters. Various visual devices can be used to display graphically the co-variation of weather elements and morbidity levels. Tromp illustrates some of these in his discussion of biometeorological

logs.¹⁷ Recent advances in computer plotting, however, render these exercises obsolete for most purposes.

Morbidity data from hospital admissions is a popular choice of morbidity index, but as was discussed in section three, this source is open to many known and also unknowable biases. In addition, the admissions represent only acute episodes of morbidity; there is usually no indication of the degree of discomfort experienced and sub-acute morbidity is totally unrepresented. A preferable course, obviously, is to monitor a fixed, homogeneous sample of asthmatics and to record the full spectrum of morbidity experienced over a set period of time.

The perception of illness is usually linked to the manifestation of, or a variation in, a particular symptom(s). In the case of asthma, the important symptoms are wheezing and dyspnoea.* A scale devised by Tromp and Limberg¹⁸ can be used to measure these symptoms. These can be recorded in a daily diary to facilitate collection and coding. The use of this scale has the added benefit that it can be used by the subjects themselves (or, as is the case in this study, by the parents of asthmatic children) to rank the severity of their attack. Furthermore, its standard use will facilitate international comparisons. It consists of a five point scale. Episodes are ranked as follow:

- | | | |
|-----|---|---------------------|
| | 0 | no complaint |
| | 1 | wheezy |
| | 2 | slightly breathless |
| | 3 | breathless |
| and | 4 | very breathless |
-

* Dyspnoea - Difficulty of breathing: breathlessness

The use of these categories allows both absolute incidence of wheezing and relative severity to be analysed. Milder episodes, for example, may possess different meteorotropic responses to those shown by acute attacks.

Analysis can proceed both by structure and by different techniques. A structural approach implies the replication of analyses for the several sub-populations that give structure to the data. For example, cross-correlation analyses can be conducted for the sexes, geographic areas, or seasons, separately or in combination, to assess the differences in morbidity due to each of these factors. Initial analyses should probably include various forms of correlation analysis for different time periods, for example, annual, seasonal, and peak events. Each of these approaches should then adopt cross-correlation analysis with the weather elements lagged on the morbidity series. The number of lags that can be usefully examined is, of course, a product of the length of the record. As in the correlations between current values, the significance of cross-correlation coefficients may be limited to a finite time-period.

The weather and morbidity data are in the form of time-series. An obvious objective of analysis is to separate the random (noise) and regular (signal) parts of the time variability in the two series. Having removed the noise, the two signals can then be compared for co-variation. Perspective, here shifts from the domain of time to that of frequency. The detection of periodicities and the estimation of the relative contribution of each period to the variation in a given series is termed "spectral analysis".

When extended to the simultaneous comparison of the two series' co-variance in the frequency domain, it is called "cross-spectral analysis". Time averaging and variable time-lags that completely obscure interrelations in the time domain using orthodox correlation methods can be usefully studied in this way. Cross-spectrum studies allow the correlation of lagged responses even when phase-shifts exist in the data. Munn advises:

"In all cases, a recommended approach is cross-spectral analysis of daily medical data with daily averages of air quality and of meteorological elements or indices; this method has been used rarely, if at all." 19

4.3 The Study Design

Introduction

Indirect data from the spatial analyses of section two and the more direct data from the temporal correlations in section three pointed to the need for detailed follow-up studies of the relationship between asthma morbidity and atmospheric quality. Important considerations in the planning were to standardise procedures and populations as far as possible to enhance both the reproducibility and the comparative value of the analysis. To this end, available standard methods have been adopted and applied to Tasmanian populations to furnish material for international comparisons. Additional to and built upon this basic exercise were innovative features and techniques aimed to contribute deeper insights to the dynamic interaction between asthma morbidity and the atmospheric environment.

The Asthma Samples

In order to obtain realistic estimates of morbidity fluctuations over time it was necessary to monitor fixed samples of asthmatics. Furthermore, the samples were required to be as homogeneous as possible in terms of age, sex and residence, etc. Homogeneity of this order was considered to be attainable only in child populations. The first requirement was to ascertain the various sources of data upon child asthmatics in Tasmania. Two have already been utilised, but for various reasons were unsatisfactory for prospective studies. Hospital morbidity statistics were highly suspect and although children's attacks

could be looked at separately there were no gradations of attack or case history details available. Similarly, the children in the 1961 birth cohort were thought to be unsuitable both because of their age, fifteen plus in 1976, and because of the increased possibility of their migration during the span of the study period when they would be leaving school.

Having discarded these two options, four potential methods of contacting young asthmatics were considered. These were:

- (a) direct population survey
- (b) general practitioner survey
- (c) child health services (aged 0-5)

or,

- (d) school health services (aged 5-15).

Given unlimited resources, the first method would have involved a large randomised survey of several hundred households to detect a suitable sample of wheezers. If a one-year age stratum was desired the sample would have had to have been enormous to obtain a reasonable number of subjects. The logistics of the survey and the superfluous collection of data from subjects not to be included in the monitored population severely reduced the viability of this approach. The next alternative offered a short-cut by tapping general practitioners' existing records. Problems encountered in regard to this included confidentiality restraints, bias in diagnosis and socioeconomic status between practices and practitioners and again the need to use multiple practices to gain a large enough sample for a given age stratum. This alternative was also rejected.

The remaining options, c and d, both appeared to be viable, and were given serious consideration. Child Health Services supply, in over 100 clinics throughout the state, medical examinations, child-rearing advice, immunisations, etc., to any who request the services. Unfortunately, the use of this service showed enormous socioeconomic and hence geographic variation. Also, the approximately ninety per cent attendance for the first major examination at six months quickly decreased to forty per cent at the age of three.²⁰ Clinic attendance records were examined for 1973-1976 and proved to be most unsatisfactory; very few of the children who attended the clinics were classified as asthmatics or wheezy-bronchitics. Over the three years only fifty-one cases were detected, sixteen on the Northwest Coast, twenty-four in Hobart and eleven scattered over the remainder of the state. The idea of using Child Health records as a source of asthmatics was, therefore, abandoned.

School Health Services' central office located in Hobart had been the centre of the original Asthma Survey conducted in 1968. Upon first entering the public, and the majority of the private schools every child is medically examined by a school medical history is taken. This information is then sent to Hobart for processing. Health records maintained for the duration of the school years are continually updated by change of residence, etc., in the central files. Permission was granted by the Minister of Health for these records to be reviewed and for names and addresses to be extracted under the supervision of the Head of School Health Services. Fortuitously, this was one of the

original investigators and the major administrator of the 1968 Tasmanian Asthma Survey, Dr H.B. Gibson. Her unwaning enthusiasm and co-operation were invaluable. Access was given simultaneously to the most recently known addresses of members of the 1961 cohort and to other more recent data upon other populations.

The team of school medical officers was briefed upon the observation of respiratory signs and wheezing in the new intake of children and as the records came in from the schools they were clerically sorted and all possible "asthma" cases were made available for scrutiny. Newly arriving in school in 1976/77 was the 1971 cohort. It was this population that was to form the basis of morbidity monitoring during their seventh year, 1977/78. The name and address of every child born in 1971 who had or had had a wheezy history was recorded. These children, a decade younger than those of the 1968 survey, formed the youngest sample of asthmatics that was available. The appropriate children's records were extracted and the parent's or guardians were contacted first by letter under the auspices of the School Health Services.

A totally unbiased sample was impossible to obtain with the available resources. The school children listed from the School Health Services were considered to be the best data obtainable at a state-wide scale. The only point of doubt was the varying physicians' diagnoses of asthma necessary for the condition to be noted on the record sheet. However, as the analysis did not concern so much the prevalence of sufferers but the incidence of episodes; local diagnostic variability did not seem to be important.

What the diagnostic criterion for detection did ensure was that the sample was of fairly well-established, unequivocal asthmatics. The spectrum of asthmatics in the sample was skewed toward the severe end. The type of mild, occasional wheezers detected by the 1968 survey would not have been defined as asthmatic and therefore, would not have been recorded on the health record as such. This, in a way, increased the sample's homogeneity. It had been intended to take a random sample of the asthmatic population. After a pilot study in one Hobart suburb it was determined that the proportion of remissions and migrants was so high as to reduce severely the gross numbers. Age-specificity was regarded as more important than randomisation, so the total population of asthmatics were drawn for each study area.

The Study Areas

Ideally, it would have been rewarding to monitor the total wheezy population in a given age-specific stratum across the entire state. In the seven-year-old stratum this would have involved the surveillance of between 1000 and 1500 persons depending on the size of the cohort. These numbers were not unmanageable but the logistics involved in the prospective study of this number of children dispersed over the island were daunting. Because of the limitations of resources in terms of finance, personnel and time, it was necessary to sample; to limit the study to particular areas of interest.

Selection of the study area was governed by the spatial patterns found in the prevalence of asthmatics in the 1961 cohort.

In particular, it was shaped by the discriminant analysis at the end of section two. A study area had to satisfy several criteria in addition to having exhibited spatial clustering in the 1961 cohort. These criteria involved accessibility, absolute numbers, the meteorological network and environmental contrast. Again, because of limited resources, it was necessary to restrict sample sites to areas of high population density. In other localities the samples would have been very small and widely dispersed and the environmental variability quite high. In addition to the need for a closely-residing, large sample of asthmatics, the location of a major meteorological station was also required. Many of the smaller settlements' stations produced very little data upon the weather other than rainfall and maximum and minimum temperatures. Lastly, the different sites were desired to be as environmentally contrasted as possible without affecting the other selection criteria.

The three broad areas of interest, highlighted by the analysis from sections two and three were the Northwest Coast, the Midlands and the Hobart Metropolitan Area. Each of these areas was outstanding for some spatial pattern of asthma morbidity and deserved detailed investigation on this criterion alone. Within these large areas, specific population clusters had to be selected. This was for three reasons: to increase environmental homogeneity, to facilitate access and to coincide clusters with meteorological stations. On the Northwest Coast both major urban centres were selected because of their population concentrations and their local site-contrasts within the same physico-climatic region. In

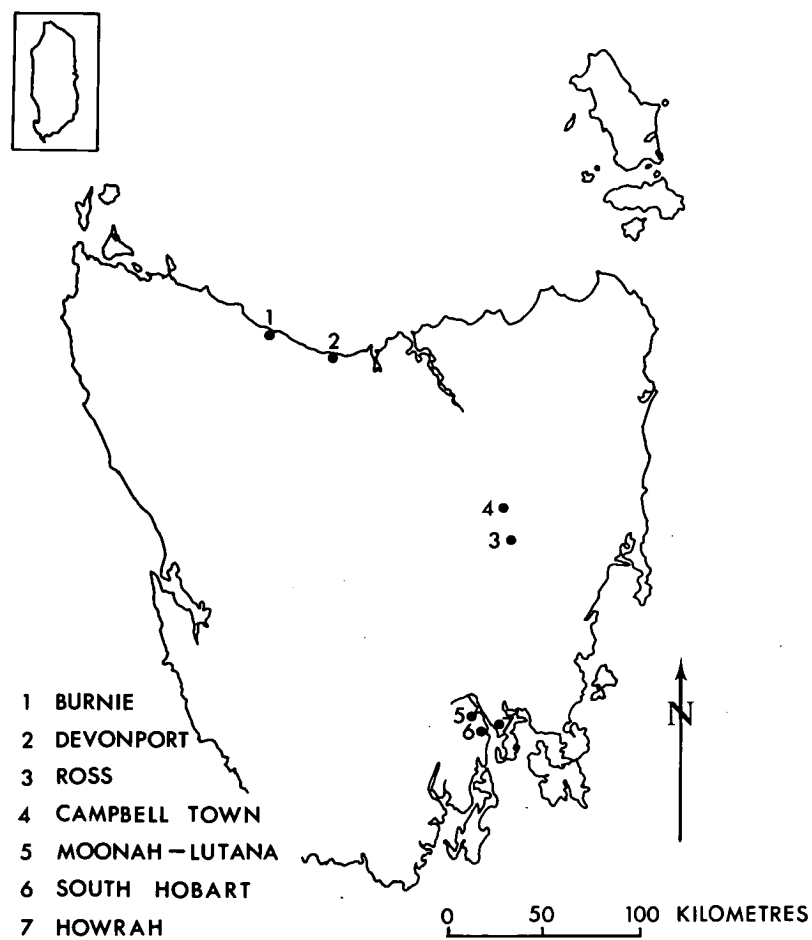


Figure 4.3.1 Location of Asthmatic Samples

the Midlands, again, the two major population nodes were selected to obtain the largest and most compact sample from this region. In the Hobart Metropolitan Area three clusters were chosen; one in close proximity to the weather bureau, one in the most heavily industrialised centre of the city and one on the Eastern shore in an area of climatic contrast to the rest of the city.

Figure 4.3.1 illustrates the location of each of these areas.

Area 1

The first of the Northwest urban centres, Burnie, is an important port and market centre but its major investment is in secondary industry. It houses large pulp and paper mills, chipboard mills, food processing plants, brick works, a sulphuric acid plant (recently closed in July 1979), and several other light industries. Burnie has a popular reputation as an asthma "black spot". This notoriety seems to be well-deserved. Burnie is backed by hills. In Summer, under a series of stagnating anticyclones, the resultant inversion conditions and an afternoon sea-breeze can increase the fumigation from factory effluent until there is a genuine threat to health.

Devonport, the second Northwestern town, an hour's drive to the East, is far less industrialised. At the heart of a rich agricultural and orchard district, its secondary industries are oriented to the processing of agricultural produce, can-making and textiles and carpets. This centre, like Burnie, is prone to Summer Easterlies. These winds are universally described by locals and visitors alike as "muggy" and "irritating", generating lassitude, migraines, headaches and accidents.

The spatial distribution of the asthmatic children within each of the sample areas is given in a series of maps in Appendix 2 (Figures A.2.1 to A.2.3). These illustrate both current wheezers and those who are enjoying a remission of their symptoms. No map is given for the Midlands' sample as these children are so few and so closely clustered that they form only two points on a map of suitable scale for this thesis format. The maps are intended to be purely descriptive. There is no analysis of variation in prevalence at the microscale and, indeed, most of the maps give no evidence of clustering that would demand further investigation.

An exception is the map of Burnie asthmatics (Figure A.2.1). Here there is a very evident clustering of active asthmatics in the hill top suburbs to the South of the Australian Pulp and Paper mills (A.P.P.M.). Asthmatics to the East and West are generally in remission. This cluster is in an area that is frequently fumigated by smoke from the A.P.P.M. (see Figure 4.4.2). The suburbs to the West are rarely affected by this type of pollution. This pattern would upon first examination appear to be indicating the culpability of pollution from the A.P.P.M. Confounding factors exist, however, in that the inhabitants of these suburbs are of low socioeconomic status and many of the asthmatic children's parents work at the factory. The suburbs to the West and East are of higher status. To what extent the cluster is due to social factors or to pollution exposure is debatable.

Area 2

Moving next to the Midlands, the centre of the state enjoys a mild "continental" effect. This is accentuated by the föhn-type winds that are caused by the Northwesterly flow of air across the state being adiabatically warmed as it falls from the central highlands. Campbelltown and Ross, only eighteen kilometers apart, each have less than one thousand population. Their economy is pastoral grazing and their only industry is that provided by saw mills. They were selected for study, even though their numbers of asthmatics were known to be low, because of their rural, inland nature that contrasted the coastal, urban makeup of all the other study areas.

Area 3

To the South, Hobart, the state capital, is dominated by government administration and high-level service functions. Nevertheless, it serves as a market centre for its peripheral primary industries, notably market gardening and orcharding and supports several secondary industries. Many of these, including one of the largest zinc smelters in the world, are located in and around the industrial park at the heart of the urban fabric. Within the Hobart Metropolitan Area, three clusters are included. These again are based upon differences shown by previous cartographic analysis. Howrah-Tramere, an Eastern shore suburb, is of interest because of its consistently different patterns to the Western shore suburbs in the analysis of the 1961 cohort. This suburb is a new growth residential area characterised by a predominance of young families. It enjoys a warmer, sunnier

climate than the Western suburbs which are dominated by the presence of Mt Wellington. Of probable influence in the Winter is the Westerly flow of urban-modified air across the river towards the Eastern shore. Almost directly West from Howrah, is the second sample area of South Hobart/Sandy Bay. This area is centred upon the Hobart Regional Meteorological Bureau and includes the University within its boundaries. Land use is basically residential and light-commercial. Like Howrah, this area is to the South of the blocking effect of Mt Wellington and is, therefore, open to the cleansing effects of the sea winds.

Further North up the Derwent Valley is the middle of the urban fabric of Hobart. Here is located the third sample area of Moonah-Lutana. This area includes most of Hobart's industrial park. Isolated from the direct influence of the sea, it is subject to the highest levels of industrial/domestic/vehicular pollution found in the city (see appendix 3). Pollution levels are exacerbated during summertime by anti-cyclonic inversions and during Winter, cold-air drainage produces a different type of inversion known to the locals as the "Bridgewater Jerry". Just to the North of the industrial park a graben forms a natural collecting basin for the cold air produced under conditions of radiation cooling on the surrounding mountain slopes. The stable, cold air mass slowly channels down the estuary often producing fog at its interface with the upper air above the inversion. This phenomenon can often last to mid-afternoon. The cold and fog associated with it encourages the increased consumption of domestic fuel, the combustion products

of which, in combination with the industrial and vehicular pollution decrease further the quality of the atmosphere.

This set of study areas chose to locate asthmatic samples residing in the North, in the South and in the middle of the state. It was reasoned that these three areas should have represented a wide range of climatic experience. Regions of more extreme climatic nature were known to exist but the population in these parts was either small or dispersed. Differences in climate between the three chosen areas, furthermore, probably represented the climate range experienced by the majority of Tasmanians. These areas were also considered to be sufficiently large to modify any meteorotropic response in their respective asthmatic populations.

According to the Köppen system of climatic classification Tasmania is termed Cbf; a marine West coast climate with a cool summer and constantly moist with rainfall all through the year. A classification better suited to small regions with rugged topography and large differences in moisture is Thornthwaite's method used by Gentilli to portray the climates of Tasmania.²¹ This system is based on "precipitation effectiveness" and "temperature efficiency". Burnie, Devonport and the rest of the Northwest Coast are classified as humid-warm and Campbelltown and Ross as dry-cool. In the Hobart area, because of the influence of Mt Wellington, the Western-shore suburbs are in a moist-subhumid-cool region and the Eastern shore enjoys a dry-subhumid-warm climate. The study areas are, therefore, seen to fall into different climatic classes. In Tasmania, however, microclimatic

effects are often seen to produce local anomalies within these defined average conditions.

To give a visual comparison of the climates for the three areas climographs were constructed (figure 4.3.2). These were based upon the 86th percentiles of the monthly maxima and the 14th percentiles of the monthly minima. A greater range was obtained in this way than would have been given by the respective means. This was necessary to discriminate more precisely the maritime climates. Tasmania's small size and oceanic situation resulted in a small, average, climatic range except in its protected interior.

Two observations are immediately obvious from the climographs; the similarity between Burnie and Hobart and the dissimilarity between them and Campbelltown. Burnie and Hobart, actually share the same average annual maximum and minimum temperatures, 16.8 and 8.3 degrees Celsius respectively. This similarity in average temperatures disguises a wide variation in other elements and in microclimatic and short-term events. For example, Burnie has, on average, 1005mm of rainfall compared to Hobart's 622mm. Campbelltown has a much wider range of maximum and minimum temperatures than the two coastal stations. Its lowest monthly minimum is reached in June reflecting the response to the solar radiation budget. In the two other stations the monthly minimum is attained in July due to the moderating effect of the sea. Similarly, the highest monthly maximum is found in January for Campbelltown and Hobart and in February for Burnie. Hobart's earlier maximum is probably due to the advection of warmer continental air from the interior

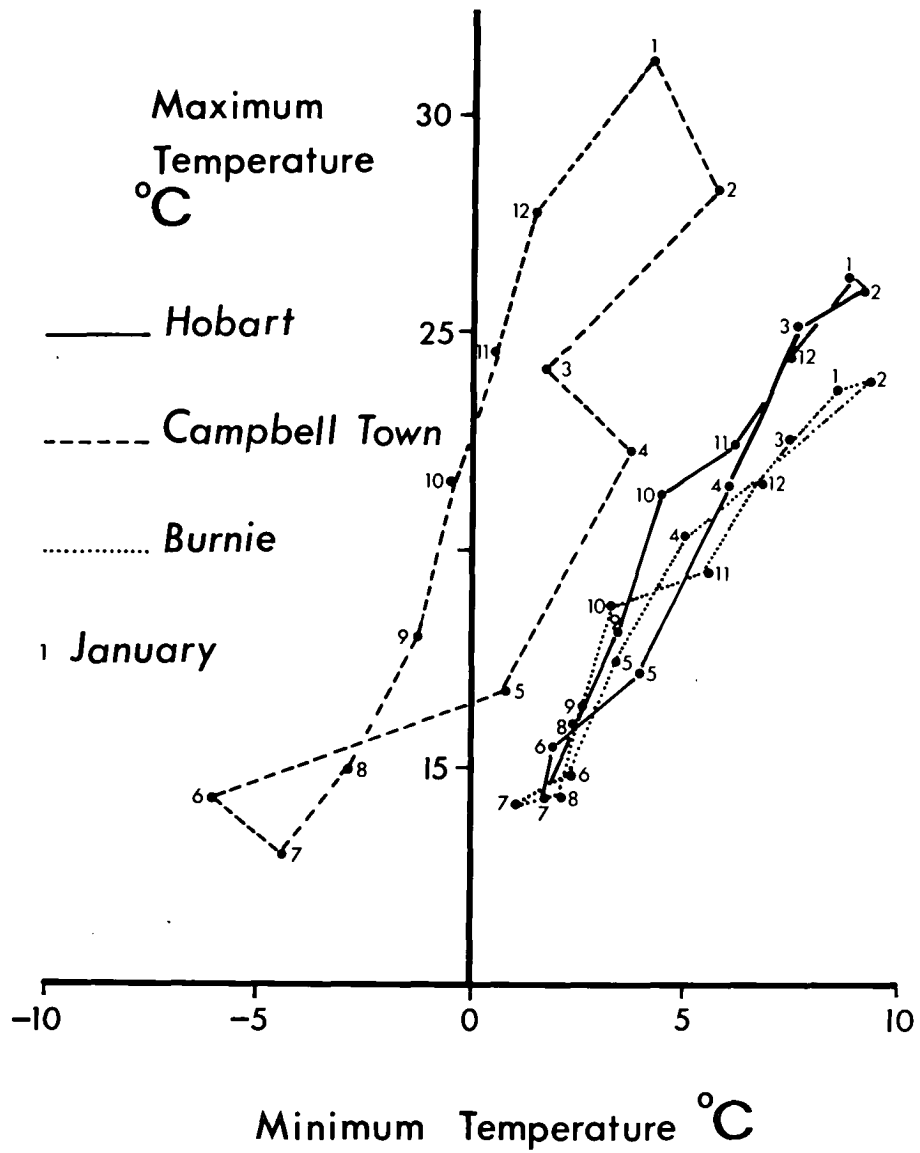


Figure 4.3.2 Climographs for the Three Major Sample Areas.

by the dominant Northwesterly winds while Burnie remains to be influenced by sea air. This effect is also signified by Hobart's higher Summer monthly maxima and increased annual range of maxima compared to Burnie.

Prospective Data Collection

The data to be collected were of two types; measurements of the physical and chemical nature of the atmosphere and measurements of the incidence and severity of asthma morbidity. These required different procedures for collection and processing.

Atmospheric Data

Most of the meteorological data were obtained from the bureau of meteorology for the nineteen month study period, 1st June 1977 - 31st December 1978. Quality varied tremendously from station to station. The Hobart Regional Office data set was perfect in the coverage of elements measured and in the non-occurrence of missing values. Campbelltown stopped as a recording station at the end of October 1978 but the records previous to this time were of fairly good quality. Burnie's data were poor. Weekends and public holidays were not recorded except for rainfall and maximum and minimum temperatures and many missing values occurred throughout the year. Hobart records existed for a large number of variables these included: 9 a.m. pressure, dry-bulb and wet-bulb temperatures, wind speed and wind direction; 3 p.m. pressure, dry-bulb and wet-bulb temperatures, windspeed and wind direction; daily maximum and minimum temperatures, rainfall, sunshine, evaporation, windrun, and the maximum gust's speed and direction.

For each station, data availability permitting, standard biometeorological indices were calculated for analyses. These included, the daily range (maximum-minimum temperatures) and the interdiurnal changes in individual elements (the products of forward-differencing). Estimates were also made of the heat-stress and cooling-power of the atmosphere for the 9 a.m. and 3 p.m. recordings. Heat stress was calculated using Thom's²² Discomfort Index,

$$D.I. = 0.4 (t_d + t_w) + 4.8,$$

where t_d is the dry bulb and t_w is the wet bulb temperature.

Cooling power was calculated by using the formula of Cena, Gregorczyk and Wójcik,²³

$$C.P. = (0.412 + 0.087 \times v) (36.5 - t),$$

where v is the wind speed in metres/second and t is the dry bulb temperature.

For Hobart only, two additional physico-chemical elements were recorded. These were atmospheric pollution and ionisation. Neither variable was regularly recorded in Tasmania. In order to measure acid-gases, which included sulphur dioxide, equipment was borrowed from the Department of Environment. The procedures involved in operating the sampling machines and the logistics necessary for the measurement of spatial variation in acid-gas levels have been discussed in detail in appendix three. Twenty-four hour, integrated, dose levels for the sampling period at the base level site in Davey Street, Hobart were coded with the meteorological data for Hobart for the same period.

The measurement of atmospheric ionisation posed greater problems. No equipment was available to borrow and the literature upon the design of ionometers was decidedly vague.²⁴ A suitable sensor had to be designed, engineered and linked to a sensitive amplifier and recorder. The history and fruition of this project have been described in appendix four. Daily fluctuations in ion levels were, like pollution levels, coded with the appropriate daily Hobart meteorological values for analysis with morbidity.

Finally, the daily synoptic charts from the Weather Bureau's Monthly Weather Reviews were examined and classified according to nine major types. These were coded thus:

- 1) anticyclone over the Great Australian Bight
- 2) anticyclone to the North of Tasmania
- 3) anticyclone over Tasmania
- 4) anticyclone over the Tasman Sea
- 5) cyclone to the West of Tasmania
- 6) cyclone over Tasmania
- 7) cut-off cyclone
- 8) stormy westerly
- and 9) zonal westerly

In addition, an estimation of the persistence of weather systems was given by coding each day with a number that represented the number of days that a particular synoptic pattern had influenced Tasmania's weather. For example, stagnating anticyclones over Tasmania were a common occurrence. In this case, the first day that a high pressure centre was over Tasmania was coded one (1) the next day

two (2) and so on until another system entered the area and displaced the high. At this point the value for persistence returned to one (1). The final atmospheric data matrix included these synoptic values with pollution, ionisation, meteorological elements and biometeorological indices.

Morbidity Data

Morbidity data upon the 1971 birth cohort samples were to be collected in two ways; cross-sectionally and longitudinally. The cross-sectional approach was by way of a questionnaire administered to the parents of the children at the start of the study. The longitudinal approach was the maintenance of a diary of morbidity as exemplified by Tromp. The questionnaire was a simplified version of that used by the 1968 Tasmanian Asthma Survey† (see appendix 1). A small pilot survey was conducted in West Hobart, a suburb not used in the major survey, to pre-test the questionnaire and to rehearse, and hence, standardise interviewing procedures. In this pre-survey parents were also asked to fill out one month's sheet of a morbidity diary in order to detect any difficulties that could be avoided by rewording written instructions or verbal explanations.

The parents of all those children living in the study areas who had had a history of asthma were contacted first by letter from the Department of School Health Services. This explained the project

†Had the standardised *Childrens Questionnaire*²⁵ been available at the time it would have been used. However, the use of wheezing on the protocol made the two questionnaires comparable in the level of information obtained.

and the impending visit from the researcher. Every household was then visited by the researcher. The questionnaire was administered and cooperation was sought to keep a morbidity diary for eighteen months. Diaries were kept for the children who were born in and who had resided only in the study area of present residence. That is, no in-migrant or out-migrant children were included in the study. The diaries were in the form of monthly sheets (see appendix 2). Parents were asked to fill these in daily and rank any wheezy attack according to Tromp's scale. The scale was explained very carefully and instructions were given to write down details for any episode about which there was any uncertainty. Columns were also provided on the sheets for the occurrence of coughs and sneezes and for additional comments, for example, the use of medication, illnesses or the suspected association of environmental events.

Families in the survey were visited regularly about every three months and the completed diary sheets were collected and additional ones given out. Three months was considered to be an optimal period for visiting. More frequent contact may have been off-putting to survey participants, while less frequent contact might have led to omissions and lack of interest. On subsequent visits, anthropometry and spirometry measurements were taken. Anthropometry included height, span, chest circumference and weight. Spirometry was measured using a Garthur Vitalograph. The best of three maximal exhalations was used to calculate the forced expiratory volumes in half-a-second and one-second, the vital capacity and the maximum expiratory flow rate. Originally, it had

TABLE 4.3.1 Breakdown of Asthma Samples by Residence and Asthmatic Status

Area	Current Wheezers	Current Remissions		Total
BURNIE	8 males* 3 females	10 males 5 females	(55.5%) (62.5%)	18 8
DEVONPORT	9 males 5 females	11 males 8 females	(52.4%) (61.5%)	21 13
MIDLANDS	5 males 3 females	1 male 2 females	(16.7%) (40.0%)	6 6
HOWRAH	11 males 4 females	7 males 0 females	(38.9%) (0.0%)	18 4
MOONAH	2 males 5 females	2 males 4 females	(50.0%) (44.4%)	4 9
SOUTH HOBART	3 males 6 females	7 males 4 females	(70.0%) (50.0%)	10 10
TOTAL	38 males 26 females	38 males 23 females	(49.4%) (46.9%)	77 49

* (+1 refusal)

been intended to collect allergy data from the subjects but resources were too limited to obtain the necessary personnel and supplies.

Table 4.3.1 gives a breakdown of the various samples by sex and morbidity status. Only one family refused to co-operate with the survey. In this case the child's status as "adopted" was the contributing factor. Of the 126 asthmatics in the sample who were born in and had lived in the same study area, seventy-seven were males and forty-nine were females. Forty-nine per cent of the males, and forty-seven per cent of the females were enjoying a remission of symptoms in 1977. The proportion of remissions varied with geographic location. Seventy per cent of the boys living in South Hobart were currently in remission compared to only seventeen per cent of the boys living in the Midlands. Sixty-two per cent of the girls from the Northwest had had remission compared to none in Howrah. Cases of remission were monitored as well as the wheezy children for the possible re-appearance of wheezy breathing and for the occurrence of cough.

At the end of the study period each child was given an identification number. For analysis each datum was an episode of wheezing. The record for each episode included the following pieces of information:

- 1) child identifier
- 2) sex
- 3) study area
- 4) date
- and 5) severity.

From this basic file the series of attacks for every sex and residence combination was processed for analysis with the meteorological data set. Eventually the meteorological and morbidity data were combined into one data file. The cross-sectional data from the questionnaire, the anthropometry and the spirometry were not strictly relevant to biometeorological study and were, therefore, included in appendix 2.

4.4 Correlation Analysis of the Relationships Between Weather and Asthma Morbidity

Introduction

Morbidity data were collected over a period of nineteen months. The first twelve months' data were analysed to establish the interrelationships between weather and morbidity in the different areas. These were to be used as the basis for modelling morbidity from weather elements. The models were to be tested subsequently for predictive skill upon the remaining seven months of data. To this end, initial analyses were to be conducted upon only the wheezy episodes experienced between the 1st June, 1977 and the 31st May, 1978.

The data were in the form of time series measured in daily increments. For each of the three study areas and their sub-areas, four series were obtained. The first two consisted of a male and a female morbidity series based upon the number of episodes of wheezing experienced on a given day regardless of the degree of morbidity experienced. The second two consisted of a male and a female weighted morbidity series. Here each episode was given a weight according to Tromp's scale. In this way, the second two series formed indices of severity of attack. Morbidity was measured not only in the first pair by absolute numbers but also in the second pair by relative strength.

All of the children were of the same age and were life-long residents of their sample areas. Homogeneity was achieved only

in regard to these aspects. For example, the use of medication varied enormously even within the same study area, (see Appendix 2). Dissimilarities were also observed in the number of attacks experienced from place to place and between individuals. It would have required, however, an impossibly large sample to control for every degree of asthmatic and medication combination. Such variability had to be accepted as one of the basic characteristics of a chronic disease.

Figure 4.4.1 illustrated the year's morbidity on a monthly basis in the form of histograms for each area by sex. Burnie's distribution was so singular that it was not combined with Devonport's episodes to form a Northwest index. Burnie's males possessed a striking Spring-Summer peak in contrast to Devonport males' Autumn-Winter peak. Burnie girls' morbidity peaked in July, December and March. In Devonport the sexes were quite similar with major peaks in April. Midlands boys demonstrated mid-Summer peaks in contrast to the girls, who had major peaks in July and minor ones in March. In Hobart both sexes peaked in Winter and males showed a smaller peak the following Autumn that was not found in females. The weighted series generally gave flatter monthly distributions; peaks were reduced and troughs were filled in. Differences between the sexes and areas gave further support to the consideration that aetiology was different for the two sexes and that environment/location factors played a part in pathogenesis. If aetiology was independent of either sex or environment then the seasonal distribution of morbidity for the sexes and the several areas would have been similar.

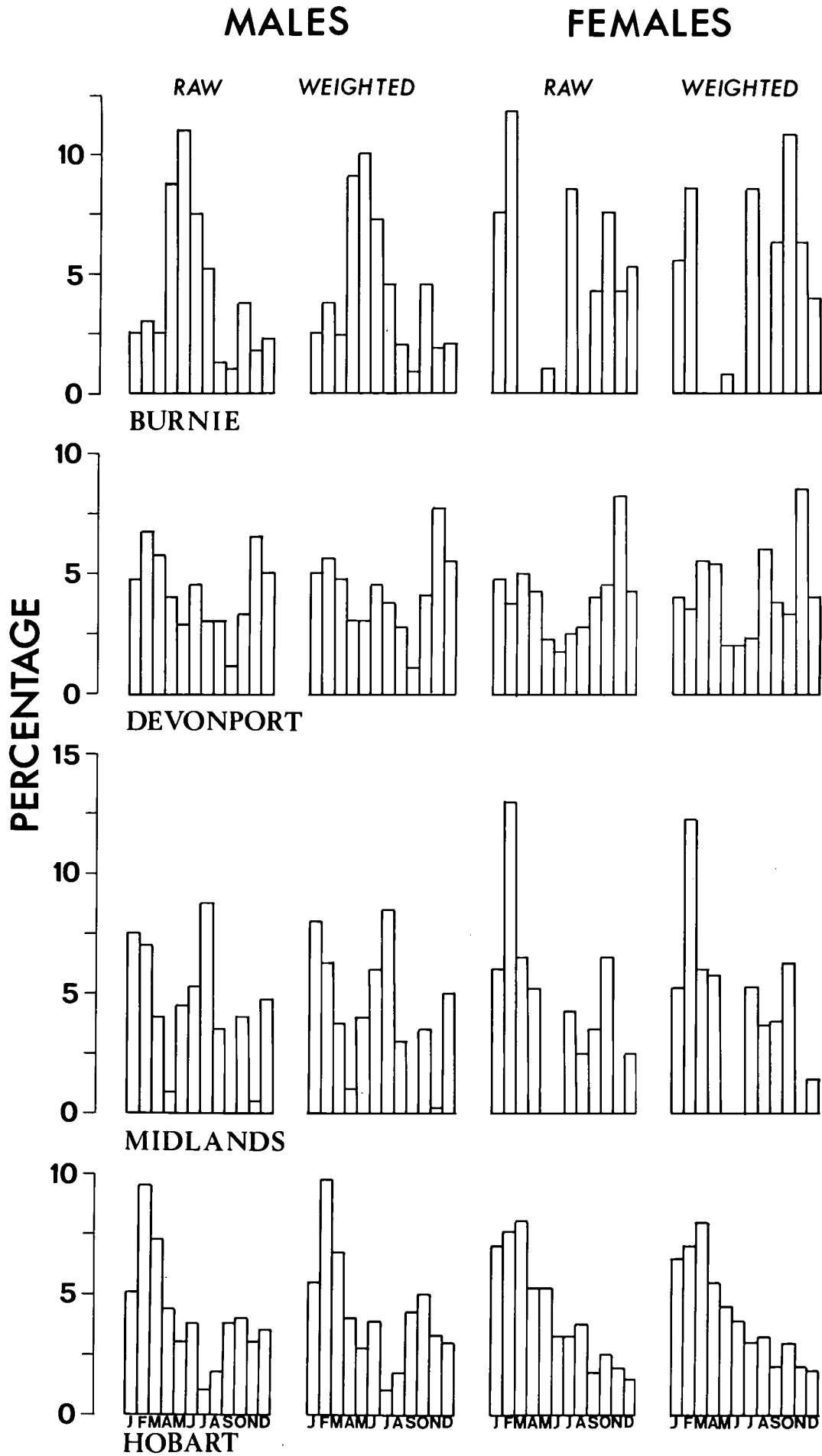


Figure 4.4.1 Percentage Distributions of Raw and Weighted Wheezing Indices by Sex and Area.

The structure of this analysis is organised along the same lines as the analysis of the hospital morbidity statistics. It starts with the full time scale and sequentially reduces to smaller events and time periods. First, the entire year's data is examined for cross-correlations with meteorological data. The time scale is then decreased to that of individual seasons and the cross-correlations are recalculated.

After the seasonal examination, small-scale weather systems are classified into the nine types described earlier and each type is dealt with separately. Cross-correlation analysis is then viewed from a different perspective. Instead of looking for one or a few fixed relationships for differing situations, an attempt is made to calculate a moving cross-correlation that portrays the changing weather-morbidity relationship over the course of the year. This gives an increased resolution to the detection of events in the continuum. Throughout the cross-correlation analyses, coefficients significant at the five per cent level and greater are usually the only ones discussed. However, in some cases where the correlations were either few or high, coefficients of lower significance were included for discussion.

Taking the year of daily values of morbidity and weather elements to be from the 1st June, 1977 to the 31st May, 1978, correlations are calculated between the weather and morbidity for each area, for both sexes separately. The results are given in table 4.4.1. Because of the large number of cases, 365 days, statistical significance is gained with a very small coefficient:

when $r = 0.085$, $p \leq 0.05$, when $r = 0.125$, $p \leq 0.01$ and when $r = 0.165$, $p \leq 0.001$. Only the correlations greater than 0.085 are given in the table. Each residence-sex category is divided further into two, a raw incidence index and a weighted severity index. Hobart, in addition to the standard weather variables available at the other stations, has evaporation, maximum gust speed, sunshine hours and pollution (acid-gas levels in $\mu\text{gm./m}^3$) measurements. Each location is next dealt with separately to enhance clarity of discussion.

i) Hobart

Even allowing for the larger number of variables included, Hobart asthmatics' annual correlations were more numerous and significant than any of the others. The correlations between weather and the severity indices of morbidity were usually slightly lower than those for the raw incidences. Generally, both sexes' morbidity had negative relationships with heat. Correlations were negative with the various temperatures and positive with cooling power. The negative correlations with temperature were higher with female morbidity indices than male morbidity indices. Males differed from females in the level of correlation and in their additional correlations with pressure (positive) and pollution (negative). Females' morbidity correlated with 3 p.m. cooling as well as the morning cooling. The highest correlation for this area was found between the raw incidence of female asthma episodes and minimum temperature, $r = -0.45$, $p \leq 0.001$. This was followed in rank by the female raw incidence

TABLE 4.4.1

Annual Correlations Between Morbidity Indices and Weather Variables:

June 1977 - May 1978

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure	12	16														
9 a.m. Dry-Bulb	-38	-35	-43	-39			-22	-17				16	-12			
9 a.m. Wet-Bulb	-33	-29	-44	-41			-21	-16				19				
9 a.m. Wind Speed															-9	
9 a.m. Cooling	38	35	43	39			14	13	11			-15				
3 p.m. Pressure	11	15														
3 p.m. Dry-Bulb	-22	-20	-32	-29			-19	-14								-9
3 p.m. Wet-Bulb	-23	-20	-39	-37			-19	-15								
3 p.m. Wind Speed	-11	-15							11						-9	
3 p.m. Cooling			23	20			16	13	10			-12				
3 p.m. Heat Stress	-23	-21	-36	-33			-19	-15								-9
Maximum Temperature	-25	-23	-35	-32			-22	-18				14	-21	-18		
Minimum Temperature	-37	-34	-45	-40			-19	-15	-14	-12	13	21				
Range of Temperature									12	10	-12		-17	-18		
Rainfall									-11				14	14		-9
Sunshine Hours	-11	-9	-9													
Evaporation	-28	-27	-24	-21												
Windrun	19	-23														
Gust Speed	-12	-17														
Acid-Gas Level	-12	-9														

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

correlation with the 9 a.m. dry bulb temperature, $r = -.044$, $p \leq 0.001$; the 9 a.m. wet-bulb temperature, $r = -0.43$, $p \leq 0.001$ and the 9 a.m. cooling effect, $r = -0.43$, $p \leq 0.001$. The asthmatic, atmospheric conditions described by these yearly associations were situations of low temperatures, low wind-speeds, low sunshine, low evaporation and low pollution levels. For males high pressure anti-cyclonic conditions seemed to be important. Their correlations were very similar to the females' but they had additional positive correlations with pressure and negative ones with wind speed. High pressure and low wind speed were characteristic of stagnant anticyclones.

ii) Midlands

In Ross and Campbelltown, only female morbidity indices attained significant correlations with weather parameters. These were similar to those found in Hobart for females but were lower in value. The highest correlations were found between 9 a.m. dry-bulb, wet-bulb, and maximum temperatures and the incidence of asthma episodes in females, $r = -0.22$, -0.21 and -0.22 respectively, $p \leq 0.001$. Midland males' correlations possessed the same signs as their Hobart counterparts but the coefficients did not reach significant levels.

iii) Northwest Coast

Burnie and Devonport had few significant correlations and the two places varied in these. Burnie male morbidity was positively correlated with 9 a.m. and 3 p.m. cooling, 3 p.m.

wind speed and temperature range and negatively correlated with minimum temperature and rainfall. The highest correlation was -0.14 with minimum temperature, $p \leq 0.005$. Female asthma severity obtained more correlations than the raw incidence did. These correlations were positive with 9 a.m. temperatures and negative with 9 a.m. and 3 p.m. cooling power and temperature range. Here the sexes seemed to have demonstrated opposite responses. Girls' attacks were related to narrow-range cooling effects. In Devonport, male morbidity was related to the cool temperatures and narrow temperature range often associated with rainfall. Female episodes were associated with very calm conditions; low afternoon wind speed, temperatures and rainfall.

Seasonal Correlations

Contrasted with the results of the hospital morbidity analysis, these correlations at the annual level of aggregation were encouraging. Analysis continued by taking each season separately. Winter was based upon the data from June 1st to August 31st. Spring was from September 1st to November 30th. Summer was from December 1st to February 28th and Autumn from March 1st to May 31st.

1) Winter

The cross-correlations between the current values for morbidity and weather elements were recorded in table 4.4.2, for the Winter period. Each location and sex specific morbidity category possessed two coefficients, one based upon the raw

incidence of wheezing, the other based upon the incidence weighted for severity. These pairs of correlations were always of the same sign, they differed only in degree. The weighted coefficients were usually, but not always, higher than their unweighted counterparts. For reasons of similarity, space and the increased amount of information from weighted data, only the coefficients with weighted morbidity were tabulated.

Hobart boys possessed their highest raw incidence correlation with 9 a.m. humidity, $r = 0.3$, $p \leq 0.005$. This value increased when weighted morbidity was used, $r = 0.36$, $p \leq 0.001$. Other correlations seemed to describe cold, damp, perhaps foggy mornings possibly achieved by cold air drainage and inversion conditions. This scenario was supported by the associations with low afternoon wind speed, and low evaporation. Hobart girls' sole significant correlation was with rainfall. Also a correlation of -0.15 , $p = 0.08$, was found with 9 a.m. pressure. Although this correlation with low pressure was of low significance, the association between female morbidity and Winter cyclonic rainfall was seen to deserve further attention.

In the Midlands, the boys' highest correlations were between 9 a.m. and 3 p.m. pressure and weighted morbidity, $r = 0.23$ and 0.22 respectively, $p \leq 0.02$. Their only other significant correlation was with 9 a.m. cooling power, $r = 0.18$, $p \leq 0.05$. Females possessed the same correlation with 9 a.m. cooling power and also a negative correlation with maximum temperature. These correlations were probably related to cold

TABLE 4.4.2

Winter Correlations Between Morbidity Indices and Weather Variables:
June 1977 - August 1977

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure						23										
9 a.m. Dry-Bulb		-18														
9 a.m. Wet-Bulb																
9 a.m. Wind Speed																
9 a.m. Cooling		18			18		18	19								
3 p.m. Pressure					17	22										-17
3 p.m. Dry-Bulb																
3 p.m. Wet-Bulb		17	18													
3 p.m. Wind Speed		-19														
3 p.m. Cooling		-20														
3 p.m. Heat Stress															-17	-22
Maximum Temperature							-18		18				20	22		
Minimum Temperature											21	21			26	18
Range of Temperature											-25	-25			-19	
Rainfall			29	28					-18							
Sunshine Hours																
Evaporation	-18	-29														
Windrun																
Gust Speed																
Acid-Gas Level																

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

Winter days, that started with a chilly morning and maintained low temperatures throughout the day. This could have occurred on days when air from the central plateau was advected Eastwards into the Midlands.

On the Northwest Coast the towns of Burnie and Devonport demonstrated remarkably similar correlations despite their different seasonal distributions shown in figure 4.4.2. Male morbidity was correlated with maximum temperature and in Burnie was, furthermore, negatively correlated with rainfall. Positive, but insignificant, correlations were also possessed between morbidity and diurnal temperature range, $r = 0.14$, $p > 0.1$. The positive correlation with maximum temperature and negative correlation with rainfall in Burnie morbidity suggested that domestic/urban pollution may have played a role in asthma exacerbation. High Winter maxima would have been achieved under anti-cyclonic stagnation conditions. Such times would have been prone to inversion and its consequent concentration of gaseous and particulate effluents. Female correlations were decidedly different to those of the males, but were again similar for both towns. Female morbidity was correlated with high minimum temperatures, low temperature range, low 3 p.m. pressure and low 3 p.m. heat. In a coastal location this correlation constellation obviously described the advection of sea air over the coast, possibly by a cyclone passing across the Bass Strait.

11) Spring

Hobart males' correlations were fairly similar in Winter and Spring. The morning correlations were almost identical with the addition of a negative correlation with minimum temperature, (see table 4.4.3). Wheezing on Spring mornings was associated with low minimum temperatures, high relative humidity, low dry-bulb temperature and high cooling power. Afternoon cooling that had been important in Winter was no longer significant. Instead, low sunshine and high rainfall gained in effect. Female morbidity, which in Winter had been associated with rainfall, was related instead to afternoon cooling power. Episodes of wheezing in Hobart females were related to low 3 p.m. dry and wet-bulb temperatures and high 3 p.m. cooling power.

Midlands males' morbidity in this season correlated positively with maximum, minimum, morning and afternoon temperatures and the 3 p.m. heat stress. This may have reflected an effect of unaccustomed heat loading on a wheezy population that was acclimatised to the cold of Winter. Females were almost exactly opposite in their correlations. They possessed negative associations with 9 a.m. temperatures, 3 p.m. wet-bulb and the maximum temperature. In addition, a positive correlation was found with 3 p.m. pressure. In Winter the sexes' relationships with weather elements were very close. By Spring, the two sexes had diverged and possessed different associations.

The close parallels between Burnie and Devonport morbidity in Winter were not evident in Spring. Burnie male morbidity was

TABLE 4.4.3

Spring Correlations Between Morbidity Indices and Weather Variables:
September 1977 - November 1977

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure																
9 a.m. Dry-Bulb	-21				26	21	-36	-34								
9 a.m. Wet-Bulb		-17			30	20	-33	-32								
9 a.m. Wind Speed					20	22										
9 a.m. Cooling	21															
3 p.m. Pressure							21	21	20							
3 p.m. Dry-Bulb			-18	-17		17										
3 p.m. Wet-Bulb			-19			17	-17									
3 p.m. Wind Speed																
3 p.m. Cooling				20					21	24		-17				21
3 p.m. Heat Stress			-19	-17		17			20							
Maximum Temperature						19	-30	-29							-17	-18
Minimum Temperature	-20					20							25	34		
Range of Temperature													-28	-28		
Rainfall		25											34	35		
Sunshine Hours	-20	-25														
Evaporation																
Windrun																
Gust Speed																
Acid-Gas Level																

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

correlated with afternoon pressure and cooling power. Burnie girls, opposite to the boys, possessed a negative correlation between their morbidity and afternoon cooling. Devonport girls' morbidity, on the other hand, had a positive correlation with afternoon cooling and was, therefore, negatively correlated with maximum temperature.

iii) Summer

Weather-morbidity correlations, (see table 4.4.4) were few in Summer. Hobart male weighted morbidity was significantly correlated with pressure. The more severe attacks that occurred at this time were probably associated with the decreased atmospheric quality related to stagnating anti-cyclones and their associated inversion conditions that would have concentrated pollution and bushfire smoke. Hobart females gave no significant correlations but, like the males, had negative correlations, $r = -0.16$, with morning temperatures. Cool mornings in Summer would have been associated with high pressure systems.

In the Midlands, male morbidity was significantly and negatively correlated with minimum temperatures and rainfall. These associations probably described two separate effects. The high morbidity with low maximum temperature could have been related to cool nights after summer warmth; an effect of cold stress during a period of acclimatisation to heat. Rainfall in a predominantly pastoral-grazing area would probably have acted as an allergen scourer, washing pollen, spores, smoke and dust from the air.

TABLE 4.4.4

Summer Correlations Between Morbidity Indices and Weather Variables:
December 1977 - February 1978

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure	21															
9 a.m. Dry-Bulb																
9 a.m. Wet-Bulb																
9 a.m. Wind Speed																
9 a.m. Cooling															-19	-19
3 p.m. Pressure		19							23	27						
3 p.m. Dry-Bulb																
3 p.m. Wet-Bulb																
3 p.m. Wind Speed																
3 p.m. Cooling																
3 p.m. Heat Stress																
Maximum Temperature									-23				-52	-51		
Minimum Temperature					-17	-19								-19		
Range of Temperature													-34	-32		
Rainfall					-23	-20										
Sunshine Hours																
Evaporation																
Windrun																
Gust Speed																
Acid-Gas Level																

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

Burnie boys' morbidity was similar to Hobart boys' morbidity. As in Hobart, female correlations were non-existent. This supported the suggestion that inversion conditions could have concentrated pollutants and increased respiratory morbidity. Male morbidity was correlated to 3 p.m. pressure and the correlation increased for weighted morbidity, $r = 0.23$ and 0.27 respectively where $p \leq 0.01$, and was, therefore, of more importance to severe attacks. Furthermore, male morbidity was negatively related to maximum temperatures. Low maximum temperatures in Summer under anti-cyclonic conditions and in coastal areas are achieved by sea breezes.

The combined effect of inversion conditions and a sea breeze are particularly important in Burnie. The town is located in a bay and is boxed in between two headlands and backing hills, (see figure 4.4.2). Under inversion conditions a lid is effectively placed over this box that opens on to the sea. With land-breeze conditions this is normally not hazardous. With a sea-breeze the box is closed and fumigation results.

In Devonport, where such physical and industrial conditions do not exist, a different relationship is demonstrated. A thermal effect is evident in Devonport male morbidity, again related to low maximum temperature and hence narrow diurnal temperature-range. The sea breezes of Summer and the occasional cool nights could stimulate attacks in a warm-adapted population. Devonport girls, unlike their neighbours in Burnie, demonstrate a correlation with 9 a.m. cooling power.

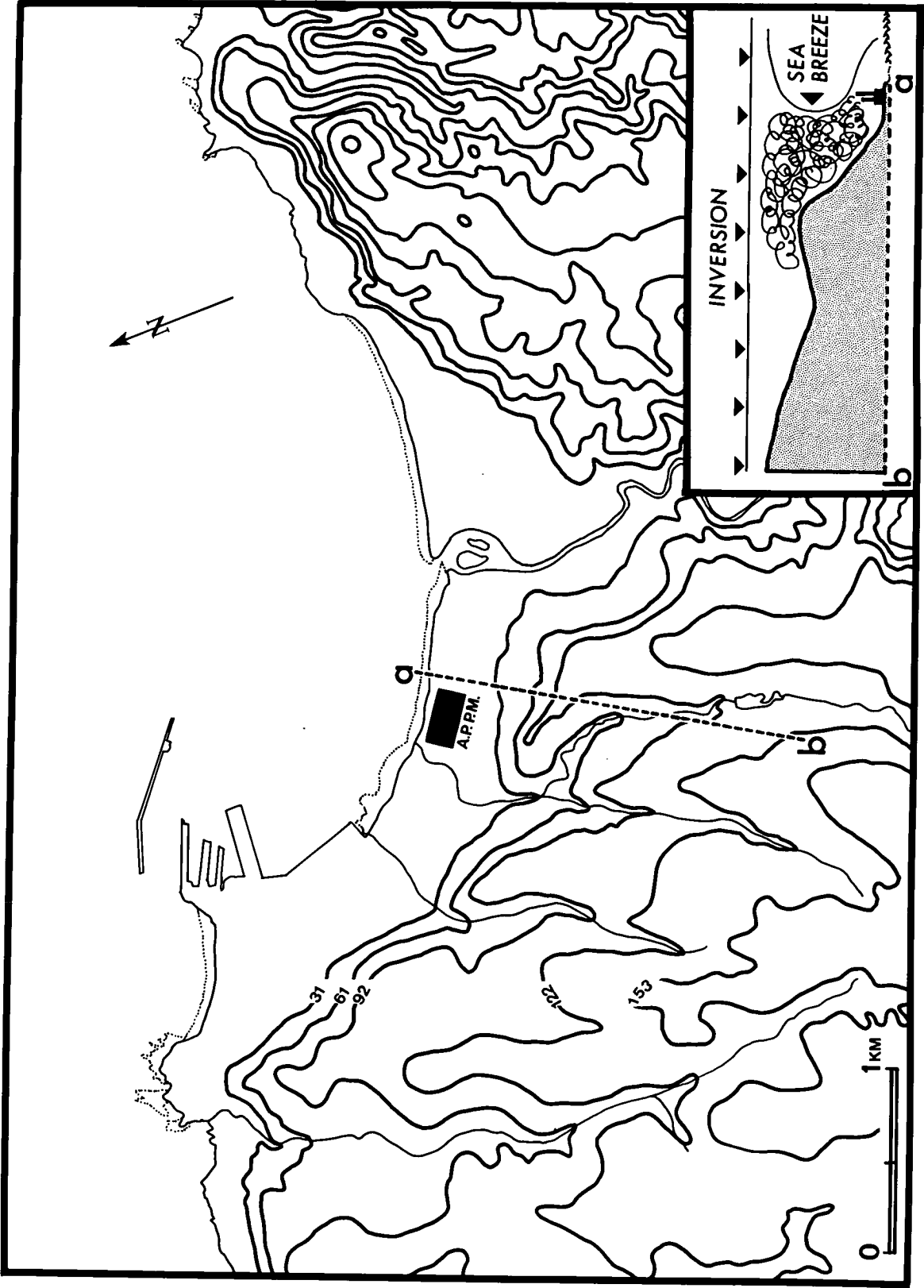


Figure 4.4.2 Burnie's Pollution Potential During Times of Upper-Level Inversions and Sea-Breezes.

iv) Autumn

In the Autumn in Hobart, males and females demonstrated very similar correlations with the weather, (see table 4.4.5). The female correlations were much higher than those for males. Only the weighted morbidity, the more severe cases of male episodes, attained significant correlations. These were negative with minimum temperature and 3 p.m. relative humidity and positive with diurnal range and sunshine. These variables were all related to high pressure centres over Tasmania that produced sunny, dry days followed by clear, crisp nights characterised by radiation-cooling.

In the Midlands too, the sexes' correlations were remarkably close. The patterns of correlation strongly resembled those for males in the Spring. Morbidity was associated with high maximum and morning temperatures. Male morbidity was also associated with low sunshine hours, and female morbidity with high maximum temperatures. It was concluded that the similarities were only apparent. Male morbidity was believed to be associated with the extensive fogs prevalent at this time of year. This would have resulted in increased minimum and morning temperatures and reduced sunshine hours. Female morbidity because of its additional correlation with maximum temperatures and wind speed was believed to be related to some other type of weather event that probably included the advection of maritime or adiabatically-warmed air into the Midlands.

On the Northwest coast, Burnie males' morbidity was correlated with the diurnal range in temperature. Devonport males

TABLE 4.4.5

Autumn Correlations Between Morbidity Indices and Weather Variables:
March 1978 - May 1978

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure																
9 a.m. Dry-Bulb					21	19	27	21								
9 a.m. Wet-Bulb					21	21	22	18								
9 a.m. Wind Speed							18									
9 a.m. Cooling											-25	-30				
3 p.m. Pressure															-17	
3 p.m. Dry-Bulb																
3 p.m. Wet-Bulb																
3 p.m. Wind Speed																
3 p.m. Cooling											-21	-24				
3 p.m. Heat Stress		-17	-19	-20							20	22				
Maximum Temperature							25	20			24	36				
Minimum Temperature		-17	-17	-18	21	18	23	19			30	37				
Range of Temperature			22	19					24	26						
Rainfall																
Sunshine Hours			27	25		-18										
Evaporation																
Windrun																
Gust Speed																
Acid-Gas Level																

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

possessed no weather-morbidity correlations of note. Burnie girls' morbidity was associated with low 9 a.m. and 3 p.m. cooling power, high minimum temperatures, high 3 p.m. heat stress and high minimum temperatures. This group of correlations could have been interpreted in several ways. They could have been due to the heat-stress associated with continental air crossing the Bass Strait from the North or warm, humid, East Australian coastal air advected by an Easterly. Alternatively, they could have been due to the ideal conditions supplied in Autumn for the growth of moulds or house-dust mites. Devonport females demonstrated only one correlation. This was between morbidity and low pressure at 3 p.m. This could have described an effect of major cold fronts coming at the end of Summer.

1-iv) The Seasons

Taken all together, one is impressed by the vast variations found between the seasons, between the sexes and between geographic areas in their morbidity-weather correlations. These findings provide more critical material to be used against the blind application of correlation analysis to hospital morbidity admissions which are standardised neither by age, nor sex, nor by residence, nor by season, nor by degree of morbidity. The benefits of standardisation are self-evident and significant seasonal differences are obvious from the correlation analyses. Questions now arise concerning the relationships found in even shorter time periods and if the seasons' meteorotropic relationships are so different, when and how do they change?

Both from the hospital morbidity analyses and now from the discussion of the seasonal patterns in this subsection, particular synoptic patterns seem to be related to increased asthma morbidity. In the following subsection these are examined for their cross-correlation with morbidity.

Synoptic Correlations

Daily synoptic weather charts had been classified into nine types as described in section 4.3. The frequency of occurrence of each type varied from twenty-six per cent to four per cent, (table 4.4.6). As well as the synoptic type the persistence of the system had to be assessed. This was based on the number of days that a given system had affected Tasmania's weather. The highest recorded length was six days. Persistence was important in the analysis of conditions when an anticyclone was stationary over Tasmania. For each type of synoptic condition, all the appropriate days were extracted and correlations were computed between their weather variates and morbidity levels. Figure 4.4.3 has been included to illustrate a typical example of each of the synoptic conditions.

i) Anticyclone over the Great Australian Bight

This synoptic pattern occurred on eighty-three days during the year, that is nearly twenty-three per cent of the time. The correlations are given in table 4.4.7. Hobart morbidity showed many significant correlations. The correlations with incidence were usually greater than those for severity. Loadings were about the same for either sex. Both had negative

TABLE 4.4.6
Frequency Distribution of Synoptic Types

	Frequency	Relative Frequency	Relative Persistence*
1. Anticyclone over Bight	83	22.7	16.8
2. Anticyclone to North	22	6.0	32.0
3. Anticyclone over Tasmania	94	25.8	28.5
4. Anticyclone to East	17	4.7	5.9
5. Cyclone over Bight	24	6.6	12.5
6. Cyclone over Tasmania	13	3.6	7.7
7. Cut-off Cyclone	20	5.5	30.0
8. Stormy Westerly	16	4.4	12.5
9. Zonal Westerly	75	20.8	19.7
TOTAL	365	100	

* Percentage persisting for 3 days or more

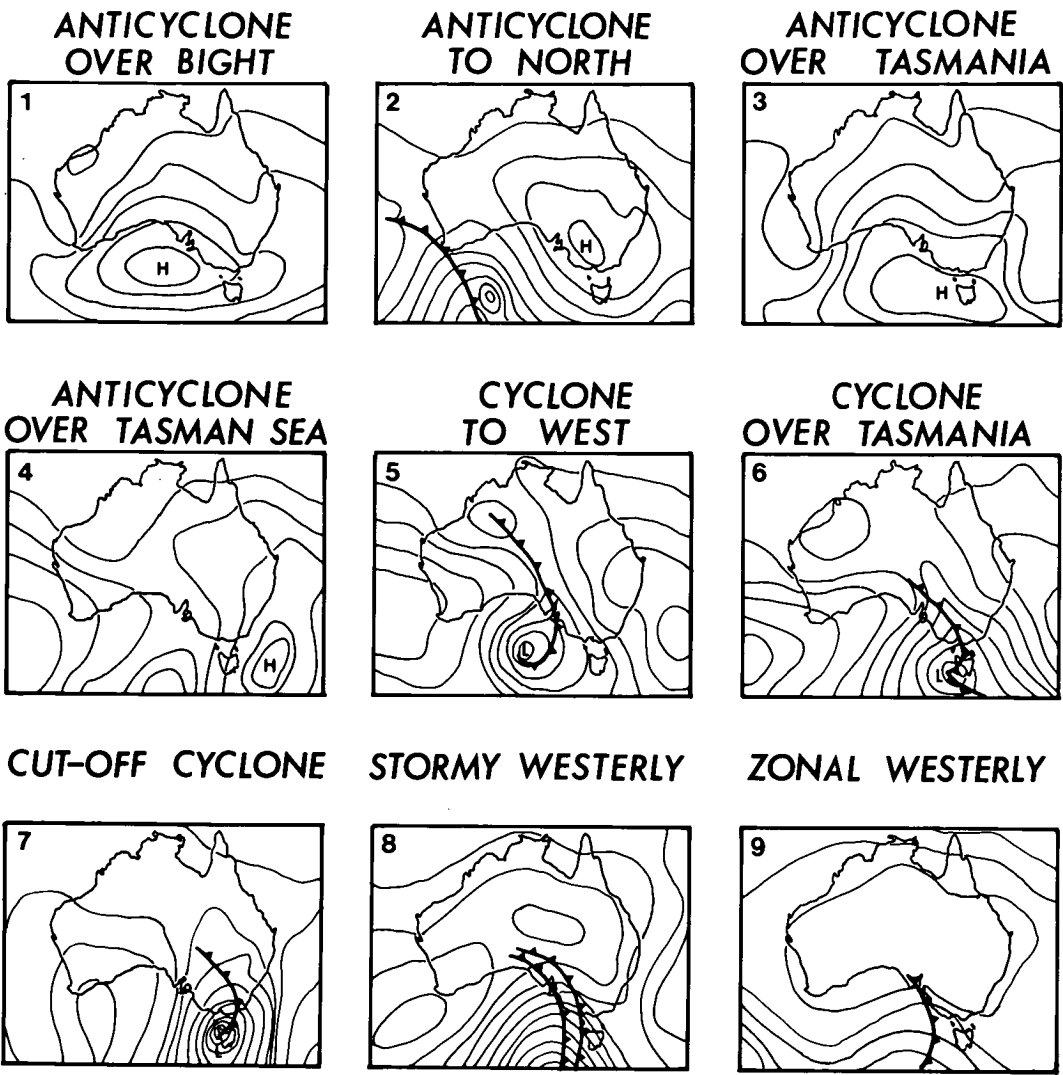


Figure 4.4.3 Examples of the Nine Synoptic Situations.

associations with temperature, afternoon heat-stress and evaporation. Females, additionally, had negative correlations with sunshine hours. Each sexes' morbidity was positively correlated with pressure, morning cooling and rainfall. Females also had significant correlations between their morbidity and 3 p.m. cooling.

In this situation, Tasmania is located on the Eastern edge of the pressure systems where winds are being advected from the South to the North. Cold, Antarctic air is brought north-eastwards across the state and forms a trough that adopts frontal characteristics. The succession of high-pressure centres is often broken by these small, single cold fronts that can bring about cold changes and rainfall.

In the Midlands boys maintained their low correlations. The female pattern was stronger for the incidence data and was close to the Hobart pattern; low temperatures and high cooling power. The correlations with rainfall were insignificant and negative.

On the Northwest Coast, Burnie males and females demonstrated different associations. Episodes in boys were positively related to pressure, wind speed, cooling power and temperature range and were negatively related to rainfall. Burnie male weighted morbidity was primarily related to afternoon heat and pressure. This was also true of Burnie females. In Devonport, males had no significant correlation between their morbidity and the weather. Female morbidity, however, was related to high pressure, low wind speed and low afternoon temperatures.

TABLE 4.4.7

Weather-Morbidity Correlations when an Anticyclone was Stationed over
the Great Australian Bight

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure	22	23		25									19	23		
9 a.m. Dry-Bulb	-36	-33	-48	-44			-21									
9 a.m. Wet-Bulb	-29	-25	-42	-41			-20					20				
9 a.m. Wind Speed									23				-24	-22		
9 a.m. Cooling	36	33	48	44					25							
3 p.m. Pressure	26	25		22					23	27						
3 p.m. Dry-Bulb	-25	-21	-39	-34						19		19				
3 p.m. Wet-Bulb	-26	-22	-32	-29						20	18	23				
3 p.m. Wind Speed									23	18			-20			
3 p.m. Cooling			40	34			20	19								
3 p.m. Heat Stress	-26	-22	-37	-33						20		21				
Maximum Temperature	-29	-26	-43	-38			-24	-20				-22	-18			
Minimum Temperature	-42	-34	-55	-50												
Range of Temperature									20							
Rainfall	33	28	38	35					-21	-20						
Sunshine Hours			-24	-22												
Evaporation	-23	-25	-30	-35												
Windrun		-18														
Gust Speed																
Acid-Gas Level																

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p \leq 0.05$ are not given.

ii) Anticyclone to the North of Tasmania

This situation occurred only six per cent of the time but when it did it demonstrated a high level of persistence; thirty-two per cent of the days had seen the system enter its third or later consecutive day, (table 4.4.8). The sexes under this synoptic condition showed very different relationships between the weather and asthma episodes. In Hobart, male episodes were associated with a narrow temperature range and high humidities and gave high correlations with pollution levels, e.g. $r = 0.64$, $p \leq 0.001$, for weighted male morbidity. Females, instead, showed the by now typical response to temperature change or cold and a positive correlation with rain $r = 0.44$, $p \leq 0.001$. This correlation may have influenced the highly negative female morbidity correlations with pollution, $r = -0.55$, $p \leq 0.001$. Rainfall would have cleansed the air of many pollutants especially soluble acid-gases.

Midlands males gave their first significant correlations observed between weather and asthma attacks. These were with high pressure, low minimum and 3 p.m. dry-bulb temperatures and increased temperature range. Females' correlations were limited to negative ones with temperature range. Other interesting but insignificant female correlations were with low maximum temperature and low wind speed, $r = -0.24$, $p \leq 0.15$ in both cases.

In the cities of Burnie and Devonport, correlations were sparse. Girls from Burnie demonstrated no relationships between their morbidity and the weather in this synoptic situation. Boys from this city had episodes associated with warm afternoons with

TABLE 4.4.8

Weather Morbidity Correlations when an Anticyclone is to the North of Tasmania

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure																
9 a.m. Dry-Bulb			-51	-49										-37		
9 a.m. Wet-Bulb			-52	-47	-35											
9 a.m. Wind Speed																
9 a.m. Cooling			51	49												
3 p.m. Pressure					45	46										
3 p.m. Dry-Bulb					-35											
3 p.m. Wet-Bulb			-36	-41												
3 p.m. Wind Speed																
3 p.m. Cooling										-44						
3 p.m. Heat Stress				-36												
Maximum Temperature				-42						37						
Minimum Temperature					-43	-38							-36	-38	39	
Range of Temperature		-35			35	36	-35	-35								
Rainfall				37					-41							
Sunshine Hours																
Evaporation																
Windrun																
Gust Speed																
Acid-Gas Level	39	64	-44	-55												

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

high maximum temperatures and low rainfall. Devonport male morbidity, however, was related to low morning dry-bulb and minimum temperatures. In an opposite fashion, Devonport girls' wheezes were associated with high minimum temperatures, $r = 0.39$, $p \leq 0.05$ and high morning dry-bulb temperatures, $r = 0.35$, $p \leq 0.06$.

iii) Anticyclone over Tasmania

This was the most frequently occurring synoptic type during the study period, 25.8 per cent, with twenty-nine per cent of its days having persisted for greater than forty-eight hours, (table 4.4.9). The basic pattern for Hobart was again that of increased morbidity with decreased temperatures and increased cooling. This was the same for both sexes. Differences between boys and girls were the positive correlations between male morbidity and pressure and the positive correlation between female morbidity and rain. In the Midlands, both sexes demonstrated the "cold effect" with an additional correlation between morbidity and 9 a.m. cooling power. There were no differences between the sexes.

Burnie males gave two correlations, both negative, between morbidity and morning wind speed and cooling power. The female morbidity associations paralleled these and included positive correlations with morning and extreme temperatures. In Burnie's environment under these synoptic patterns heat stress seemed to play a larger role than the cold stress evident in more southerly parts. Devonport boys' morbidity correlated with low diurnal range in temperature. Females in Devonport had their

TABLE 4.4.9

Weather Morbidity Correlations when an Anticyclone is Stationed over Tasmania

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure	37	38					26									
9 a.m. Dry-Bulb	-40	-34	-46	-43	-22	-21	-34	-20				25				
9 a.m. Wet-Bulb	-37	-31	-47	-44	-22	-21	-31	-18				31				
9 a.m. Wind Speed		17	18	17						-17	-25	-25				
9 a.m. Cooling					21	17	26	19	-19	-17	-27	-35				
3 p.m. Pressure	35	35														
3 p.m. Dry-Bulb	-21		-31	-33	-28	-25	-43	-27							-17	-18
3 p.m. Wet-Bulb	-23		-41	-42	-30	-28	-44	-28							-18	-2
3 p.m. Wind Speed	-24	-20	-17	-17												
3 p.m. Cooling	-40	34	46	43	18	16	35	21				-23				
3 p.m. Heat Stress	-23		-36	-38	-29	-27	-44	-28							-18	-19
Maximum Temperature	-22		-30	-32	-24	-22	-32	-17				19				
Minimum Temperature	-38	-35	-47	-43		-16	-22				16	27				
Range of Temperature		17											-19	-24		
Rainfall			24	29												
Sunshine Hours																
Evaporation	-28	-25	-24	-21												
Windrun	-20	-21														
Gust Speed																
Acid-Gas Level																

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

morbid episodes associated with low afternoon heat stress and low afternoon temperatures.

The conditions caused by stagnating anticyclones can decrease atmospheric quality and, hence, contribute to increased respiratory morbidity. Inversions are common in these situations. They retard the diffusion and turbulent mixing of gaseous effluents from their various sources and lead to localised concentrations of pollutants. The longer the inversion conditions last the more concentrated and irritating the pollution becomes. To examine these effects, the day when an anticyclone had been stationary over Tasmania for at least two days are selected for analysis. The results are given in table 4.4.10.

When tables 4.4.9 and 4.4.10 are compared the coefficients are generally seen to be increased in the latter. The basic pattern for Hobart children remains that of the "cold effect", especially in the mornings. The correlations with severity are, in this situation, usually greater than those with incidence. The highest correlations are found between the female morbidity indices and minimum temperature, $r = -0.66$ and $r = -0.71$, $p \leq 0.001$. Male correlation patterns follow the female ones at a slightly lower level. Also, males possess correlations between their morbidity and high pressure and low evaporation. Oddly, female morbidity, that in conditions of an anticyclone to the North is negatively correlated with pollution, gains highly significant positive correlations with pollution, $r = 0.57$, 0.59 , $p \leq 0.001$, while males possess insignificant negative coefficients.

TABLE 4.4.10

Weather-Morbidity Correlations when an Anticyclone is Stationed over Tasmania for at least three days

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure	40	46														
9 a.m. Dry-Bulb	-39	-33	-54	-58	-50	-51	-50		48	33	-31		50	47	38	40
9 a.m. Wet-Bulb	-39	-35	-56	-59	-51	-52	-50		42				44	41	33	37
9 a.m. Wind Speed	38	48	39	43							-31					
9 a.m. Cooling	39	33	54	57	46	40										
3 p.m. Pressure	38	43													-39	
3 p.m. Dry-Bulb	-38		-37	-43	-56	-59	-46									
3 p.m. Wet-Bulb	-41	-32	-51	-53	-59	-61	-50					-33				
3 p.m. Wind Speed																
3 p.m. Cooling					53	51	36									
3 p.m. Heat Stress	-41	-30	-44	-49	-58	-60	-48					-32				
Maximum Temperature	-33		-36	-42	-51	-51	-39									
Minimum Temperature	-39	-41	-66	-71	-45	-46	-44						37	37		
Range of Temperature									39	53					32	
Rainfall																
Sunshine Hours	-35															
Evaporation	-49	-39														
Windrun	-41	-32														
Gust Speed																
Acid-Gas Level			57	59												

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

In the Midlands, again, the correlation structure is similar to that of Hobart children, with an additional emphasis upon afternoon cooling. Boys appear to be more susceptible to morning cooling power. None of the correlations between the female severity index and the weather variables have statistical significance.

Males from Burnie and Devonport display very closely similar patterns. These are new to the relationships shown in table 4.4.9. Here, morbidity in males from Burnie and Devonport show a correlation with increased morning temperatures and the diurnal range in temperatures. Female morbidity in Burnie is related to the same correlation pattern as Hobart.

The correlations found with stagnating anti-cyclonic conditions were interesting but it was difficult to accept that Summer anticyclones would produce the same effects as Winter anticyclones. To examine this, two more correlation matrices were produced (see tables 4.4.10a and b). These exhibited correlations for June 1st to August 31st, 1977 and December 1st, 1977 to February 28th, 1978, separately for the days on which anticyclones had persisted for forty-eight hours or longer. In Summer this amounted to seven days and in Winter six days.

In Summer, conditions dominated by stagnant anticyclones produced striking correlations between weather and morbidity. In Hobart, males had far more correlations than females. These were with pressure. The highest correlation was, $r = 0.9$, $p \leq 0.005$, between 9 a.m. pressure and male incidence. Negative correlations were with wind speed, 3 p.m. heat stress, maximum

TABLE 4.4.10.a
Weather-Morbidity Correlations when an Anticyclone is Stationed over Tasmania in Summer for at least three days

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure	90	75														
9 a.m. Dry-Bulb	-50				-78	-78			81	66						
9 a.m. Wet-Bulb					-63	-63										
9 a.m. Wind Speed	-65	-77							82	70						
9 a.m. Cooling									74	64						
3 p.m. Pressure	86	74							62	79	-84					
3 p.m. Dry-Bulb	-61								64	53	-99	-99				
3 p.m. Wet-Bulb									60		-99	-99				
3 p.m. Wind Speed	-75	-59			79	79			61	84						
3 p.m. Cooling					93	93										
3 p.m. Heat Stress	-64	-42							62	52	-99	-99				
Maximum Temperature	-63	-47			55	55							-60	-60		
Minimum Temperature					-58	-58							-54	-54		
Range of Temperature	-64	-63			70	70										
Rainfall																
Sunshine Hours																
Evaporation																
Windrun																
Gust Speed			-63	-64												
Acid-Gas Level	-31	-49	33	33												

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

TABLE 4.4.10.b
Weather-Morbidity Correlations when an Anticyclone is Stationed over Tasmania in Winter
for at least Three Days

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure			-86	-84												
9 a.m. Dry-Bulb	75	83	-71					-80	73	73						
9 a.m. Wet-Bulb	74	79						-77	89	89						
9 a.m. Wind Speed	71															
9 a.m. Cooling	-75	-83	71													
3 p.m. Pressure			-92	-91											-87	-87
3 p.m. Dry-Bulb	73	78													-98	-98
3 p.m. Wet-Bulb		74	-72	-72											-97	-97
3 p.m. Wind Speed																
3 p.m. Cooling																
3 p.m. Heat Stress		76													-97	-97
Maximum Temperature	75	79														
Minimum Temperature	83	91	-78	-75												
Range of Temperature											-79	-79				
Rainfall																
Sunshine Hours																
Evaporation																
Windrun																
Gust Speed																
Acid-Gas Level																

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

temperature and range in temperature. The correlations with pollution were both negative and insignificant. Females' sole significant correlation was a negative one with 3 p.m. relative humidity.

Midlands males and females demonstrated different associations. Males had a "cold effect" pattern; negative correlations with morning temperatures and positive correlations with afternoon wind speed and cooling power. Male morbidity and 3 p.m. cooling had a coefficient of 0.93, $p \leq 0.001$. Females, although their correlations were different, demonstrated another "cold effect" pattern. Their significant correlation was with diurnal range, $r = 0.7$, $p \leq 0.05$, but this was reinforced by the existence of two high but insignificant correlations. These were a positive correlation with the maximum and a negative correlation with the minimum temperature.

In Burnie the boys and girls once more exhibited different weather-morbidity relationships. Males had positive correlations with 9 a.m. dry-bulb temperature wind speed and cooling power and with 3 p.m. pressure and wind speed. Females demonstrated a "cold effect"; they possessed negative correlations with 3 p.m. pressure, dry-bulb and wet-bulb temperatures and heat stress. The last three coefficients were all very high, $r = 0.99$, $p \leq 0.001$. In Devonport, neither males nor females gave significant correlations. The females' highest coefficient was with morning cooling power, $r = 0.49$, $p = 0.15$. Devonport males' highest correlation was with maximum temperatures, $r = -0.6$, $p = 0.08$.

During the Winter months changes took place. Hobart male morbidity that in Summer had demonstrated a typical "cold effect" pattern was now related to high morning, afternoon and extreme temperatures and 3 p.m. heat stress, low 9 a.m. wind speed and high afternoon humidity. Girls, on the contrary, presented a "cold effect" pattern; negative correlations with pressure, temperature and 3 p.m. heat stress and positive correlations with 9 a.m. cooling and relative humidity. Both sexes in the Midlands gave "cold effects". Midland male morbidity was related to wind speed and cooling power, $r = 0.69$ and 0.62 respectively. Because of the small numbers, neither of these was significant. Females' weighted morbidity in Ross and Campbelltown possessed negative correlations with morning temperatures, $r = -0.8$, $p \leq 0.05$. Their negative correlation with minimum temperature was of only low significance, $r = 0.67$, $p = 0.08$. Burnie males gave correlations that were close to those found in Hobart males; positive correlations with morning temperatures and positive, but insignificant, relationships with maximum temperatures and diurnal range. Burnie female morbidity, however, resembled Hobart female morbidity. Its significant correlation was given with diurnal range, $r = -0.79$, $p = 0.03$. Further high, but insignificant, correlations coloured this relationship. They were negative correlations with 9 a.m. pressure and the maximum temperature. Devonport boys had no significant correlations. The females from this town, like the Hobart and Midlands girls demonstrated a "cold effect" pattern. The

coefficients were negative with 3 p.m. pressure, wet-bulb and dry-bulb temperatures and heat stress. The highest coefficients were with the latter three variables, $r = -0.98$, -0.97 and -0.97 respectively, $p \leq 0.001$.

iv) Anticyclone over the Tasman Sea

An anticyclone stationed over the Tasman Sea existed only seventeen times during the year studied. Because of this small number only large correlation coefficients gained statistical significance. During these times Hobart males' only significant morbidity correlation was a negative one with pollution. Female correlations with this variable were positive but insignificant. Hobart females possessed associations with low rainfall, low humidity and increased wind and evaporation (table 4.4.11). Midland females' morbidity yielded no correlations with the weather. Midland male morbidity was correlated with 9 a.m. and 3 p.m. cooling power and, consequently, low afternoon heat stress and temperatures. Burnie children's sole significant correlation was between low minimum temperature and male weighted morbidity. An examination of the less-significant correlations showed that the boys had the typical "cold effect" and the girls a "warm effect". The Devonport children's only correlation was with 9a.m. pressure.

v) Cyclone to the West of Tasmania

In Hobart males this synoptic type produced the familiar "cold effect" correlations and an additional negative correlation with evaporation (table 4.4.12). In females from Hobart,

TABLE 4.4.11
Weather-Morbidity Correlations when an Anticyclone is Stationed over the Tasman Sea

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure															46	
9 a.m. Dry-Bulb																
9 a.m. Wet-Bulb																
9 a.m. Wind Speed																
9 a.m. Cooling								62								
3 p.m. Pressure																
3 p.m. Dry-Bulb								-42	-41							
3 p.m. Wet-Bulb								-47	-48							
3 p.m. Wind Speed																
3 p.m. Cooling								51	60							
3 p.m. Heat Stress								-45	-45							
Maximum Temperature								-44	-45							
Minimum Temperature																-40
Range of Temperature																
Rainfall																
Sunshine Hours																
Evaporation																
Windrun																
Gust Speed																
Acid-Gas Level																

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

TABLE 4.4.12
Weather-Morbidity Correlations when a Cyclone is to the West of Tasmania

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure				-36			-36	-42							36	36
9 a.m. Dry-Bulb	-47	-49						-33				-33				
9 a.m. Wet-Bulb	-48	-47						-35								
9 a.m. Wind Speed			-37	-36												
9 a.m. Cooling	47	49												-35		
3 p.m. Pressure			-35	-38												
3 p.m. Dry-Bulb																
3 p.m. Wet-Bulb	-41	-43														
3 p.m. Wind Speed		33														
3 p.m. Cooling								34								
3 p.m. Heat Stress	-35	-38														
Maximum Temperature		-34														
Minimum Temperature	-47	-49						-39								
Range of Temperature																
Rainfall			-37	-40												
Sunshine Hours																
Evaporation	-33	-38														
Windrun																
Gust Speed																
Acid-Gas Level																

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

morbidity was related to low pressure, low wind speed and low rainfall. Midlands females gave correlations between their morbidity and low pressure, minimum and morning temperature and high afternoon cooling power. Devonport girls' morbidity was positively associated with pressure and negatively associated with morning cooling power. Burnie females' had one correlation. This was between their weighted morbidity index and the 9 a.m. dry-bulb. No males, other than those in Hobart demonstrated any significant meteorotropisms. This pattern occurred on twenty-four days during the year.

vi) Cyclone Over Tasmania

A low pressure centre only rarely established itself directly over Tasmania. This only happened on thirteen days during the year. At these times the correlation matrix was rather different to that found during conditions of anticyclonic dominance. Temperature effects decreased in importance as did the number of correlations with Hobart children's morbidity. Midlands children gave no correlations at all, (table 4.4.13).

Boys living in Hobart possessed two significant correlations, a positive relationship to 3 p.m. pressure and a negative one with daily wind run. The coefficients for wind run were high $r = -0.72$ and -0.69 , $p \leq 0.07$. High, but insignificant, correlations included negative ones with sunshine and evaporation and positive ones with relative humidity. Females' correlations were directly opposite. They had strong positive correlations with 9 a.m. wind speed, daily wind run, and evaporation and a

TABLE 4.4.13
Weather-Morbidity Correlations when a Cyclone is Stationed Over Tasmania

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure															68	
9 a.m. Dry-Bulb									-52				-54			
9 a.m. Wet-Bulb																
9 a.m. Wind Speed				58	53											
9 a.m. Cooling																
3 p.m. Pressure			52													
3 p.m. Dry-Bulb																
3 p.m. Wet-Bulb				-52									48	50		
3 p.m. Wind Speed																
3 p.m. Cooling														-46		
3 p.m. Heat Stress																
Maximum Temperature													-61		-54	
Minimum Temperature														53		
Range of Temperature													-76	-65		
Rainfall													51	63		
Sunshine Hours																
Evaporation				67	45											
Windrun	-72	-69	60	60												
Gust Speed																
Acid-Gas Level			47													

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

negative correlation with the 3 p.m. wet-bulb temperature. In the incidence of attacks, females possessed a positive correlation with pollution, $r = 0.47$, $p \leq 0.01$.

Burnie girls had no weather correlations during these times and boys only one. This was a negative correlation with the 9 a.m. dry-bulb temperature. Devonport girls gave significant correlations with two weather variables, 9 a.m. pressure and low maximum temperatures. Boys living in Devonport possessed the highest number of correlations. Their largest correlation was between the incidence of attacks and range in temperature, $r = -0.76$, $p \leq 0.001$. The other correlations included negative ones with 9 a.m. dry-bulb temperatures, 3 p.m. cooling power and maximum temperature and positive ones with 3 p.m. heat stress, minimum temperature and rainfall. Depending on the configuration of the low pressure centre, this could have been due to the advection of warm humid air from the North across to Tasmania.

vii) Cut-Off Cyclone

This synoptic type occurred on twenty days in the year. It was characterised by the deformation of an approaching southern trough to a small cyclone. This was usually accompanied by heavy rain/snow and rapid change. The "cold effect" was obvious from the correlations.

Hobart males' morbidity under this synoptic process were related to low 9 a.m. pressure and high relative humidity. Female morbidity was related to high pollution levels. Females

TABLE 4.4.14
Weather-Morbidity Correlations When a Cut-Off Cyclone is Influencing Tasmania's Weather

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure	-39	-39									-41		54	39		
9 a.m. Dry-Bulb																
9 a.m. Wet-Bulb																
9 a.m. Wind Speed					50	43	62	52	40	40						48
9 a.m. Cooling							72	59	44	43						48
3 p.m. Pressure					45		57	49					45			
3 p.m. Dry-Bulb																
3 p.m. Wet-Bulb																
3 p.m. Wind Speed																
3 p.m. Cooling					66	56	48		44	47						43
3 p.m. Heat Stress																
Maximum Temperature																
Minimum Temperature																
Range of Temperature																
Rainfall																
Sunshine Hours																
Evaporation																
Windrun																
Gust Speed																
Acid-Gas Level				45												

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

possessed some additional high but insignificant correlation with 3 p.m. cooling and low sunshine. In the Midlands, both sexes demonstrated high correlations with atmospheric cooling. The highest correlation was found between the Midlands girls' incidence and 9 a.m. cooling, $r = 0.72$, $p \leq 0.001$. This cooling pattern was also demonstrated by Burnie boys and Devonport girls. Devonport boys' correlations were with morning and afternoon pressure (table 4.4.14).

viii) Stormy Westerlies

Stormy westerlies, characterised by high wind speeds and the quick succession of two or more cold fronts very close together occurred on sixteen days. Hobart children's morbidity once more gained the largest number of correlations (table 4.4.15). These followed the "cold effect" pattern. The highest correlation was between male incidence and minimum temperature, $r = -0.74$, $p \leq 0.001$. Boys had many more correlations than girls. The girls' associations were with low minimum temperature, low evaporation and low pollution levels. The correlation with pollution was high, $r = -0.68$, $p \leq 0.001$. Boys' correlations were negative with morning and afternoon temperatures, 3 p.m. heat stress, sunshine, evaporation, wind speed and pollution and positive with cooling power and relative humidity.

In the Midlands, boys' attacks correlated with a narrow temperature range. Other high but insignificant correlations were with 9 a.m. cooling power and rainfall. Female morbidity was different. All female correlations were negative. These

TABLE 4.4.15
Weather-Morbidity Correlations When Stormy Westerlies are Affecting Tasmania's Weather

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure																
9 a.m. Dry-Bulb	-58	-59					-41		48	48						
9 a.m. Wet-Bulb	-56	-59							49	50						
9 a.m. Wind Speed	-46	-45					-47	-41								
9 a.m. Cooling	58	59														
3 p.m. Pressure	-46	-42														
3 p.m. Dry-Bulb	-64	-64					-47	-45	41	41	-56	-56				
3 p.m. Wet-Bulb	-58	-61							42	42	-57	-57				
3 p.m. Wind Speed											-46	-46				
3 p.m. Cooling																
3 p.m. Heat Stress	-62	-63							41	41	-57	-57				
Maximum Temperature	-70	-70					-43	-43	44	46						
Minimum Temperature	-74	-72	-48	-44			-57		45	44						
Range of Temperature					-46	-54							-52	-43	-65	-54
Rainfall										47						
Sunshine Hours	-61	-58														
Evaporation	-57	-55	-41													
Windrun	-47	-50														
Gust Speed	53	49														
Acid-Gas Level	-62	-62	-68	-68												

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

were with 9 a.m. wind speed, 3 p.m. dry-bulb temperature, maximum temperature and the minimum temperature.

Burnie boys and girls showed very different correlations. The male correlations were all positive. They included the morning and afternoon temperatures, 3 p.m. heat stress, the maximum and minimum temperatures and rainfall. Girls, instead, gave the "cold effect" pattern of negative correlation with temperature and 3 p.m. heat-stress. Devonport children possessed only one correlation with a weather element. This was with the diurnal range in temperature. Their morbidity was associated with a narrow daily range.

ix) Zonal Westerlies

The last synoptic category is one of the more common situations that occurs for twenty-one per cent of the year. Basically, it describes the general westerly flow of the southern trough. A cold air mass with discrete frontal conditions that affect Tasmania's weather by bringing coolness and rain. The morbidity-weather correlation matrix is very similar to that calculated for stormy westerlies but the coefficients are generally smaller due to the differences in type and degree of meteorological stress produced by the two situations. The correlations are summarised in table 4.4.16. Again, the "cooling effect" is prominent in the correlations.

In Hobart the male morbidity correlation structure was very similar to that found with stormy westerlies. The coefficients were lower but the increased number of days made

TABLE 4.4.16
Weather-Morbidity Correlations when Zonal Westerlies are Affecting Tasmania's Weather

	HBR	HBW	HGR	HGW	MBR	MBW	MGR	MGW	BBR	BBW	BGR	BGW	DBR	DBW	DGR	DGW
9 a.m. Pressure	29	30	22			23	21									
9 a.m. Dry-Bulb	-55	-55	-56	-49	30						26	31	-31	-28		
9 a.m. Wet-Bulb	-48	-47	-57	-49	29						21	26	-25	-23		
9 a.m. Wind Speed															-23	-26
9 a.m. Cooling	55	55	56	49	-24								20	22		-22
3 p.m. Pressure	26	28	22			24	21									
3 p.m. Dry-Bulb	-32	-33	-40	-32	29	20										
3 p.m. Wet-Bulb	-32	-33	-46	-40	25											
3 p.m. Wind Speed	-21	-24														
3 p.m. Cooling					-24			-23								
3 p.m. Heat Stress	-33	-33	-43	-36	28	19										
Maximum Temperature	-35	-36	-47	-40	30	20					20	27	-40	-39		
Minimum Temperature	-52	-51	-53	-48	22				-25	-20	31	33				
Range of Temperature							23	26					-29	-32		
Rainfall																
Sunshine Hours	-23	-22														
Evaporation	-44	-44	-35	-36												
Windrun	-30	-33														
Gust Speed																
Acid-Gas Level	-21	-25														

COLUMN LEGEND: Hobart Boys Raw
Midland Girls Weighted
Burnie
Devonport

Decimal points have been removed by multiplying the coefficients by 100.
Coefficients not significant at $p < 0.05$ are not given.

them gain in significance. New correlations were with pressure. Female morbidity-weather correlations increased from the two found with stormy westerlies to over a dozen with zonal systems. The correlations were very close to those found with male morbidity. Female morbidity, however, lacked the male correlations with low 3 p.m. wind speed, low sunshine, high 9 a.m. relative humidity and low pollution.

In the Midlands, both sexes showed correlations that were opposite to those found in the Hobart children's morbidity. Boys demonstrated positive correlations with 9 a.m. pressure, morning and afternoon temperatures, extreme temperatures and the diurnal range in temperature. Negative correlations were found with 9 a.m. and 3 p.m. cooling power. Girls' correlations were positive with 9 a.m. and 3 p.m. pressure and diurnal range and negative with 3 p.m. cooling. During westerly conditions, warm days in the Midlands would have been accentuated by winds that have been adiabatically warmed as they fell from the central and western highlands.

Burnie and Devonport's males and females demonstrated dissimilar correlation patterns. Burnie males possessed only one correlation. This was a negative correlation with minimum temperature. Females in Burnie had positive correlations with morning and afternoon temperatures. Devonport males showed the "cooling effect" pattern of correlations and an association with a narrow diurnal range in temperature. These were strong negative coefficients with morning and maximum temperatures and positive coefficients with morning and afternoon cooling.

Devonport females, like Burnie females, did not seem to be affected by the cooling power of the atmosphere. Their correlations were negative ones with morning windspeed and cooling power.

Synoptic Summary

The correlation analyses conducted for the several different synoptic situations revealed many correlations that had not been considered previously. These coefficients were generally higher than those obtained at annual or seasonal scales and in some instances were very high indeed. This increased level of correlation suggested that the differences in the average correlations of the seasons and other long time periods may have been due to the variation in frequency and persistence of the synoptic patterns during these scales of time. It was noticed that as the time period of analysis decreased, and the weather situation was more precisely described, the weather-morbidity correlations magnified in both level and significance. This was exemplified by the detailed treatment of the periods of stagnating anticyclones. Here, coefficients of almost unity were obtained between certain weather elements and morbidity.

The most widespread meteorotropism involved with asthma morbidity was the so called "cold effect" or "cooling effect". This was postulated by Tromp over a decade ago and has been firmly corroborated by these analyses. The extent and degree of this effect, however, appeared to vary between the sexes, between the places and between synoptic patterns and different time

periods. It was possible that the different populations' "morbidity limit" and hence, sensitivity to meteorological stress were a product of acclimatisation and past stimuli. It remained to be seen how a given meteorotropism, found in isolated synoptic events, behaved in the time continuum. To examine this facet of the relationships between weather and morbidity, moving correlations were calculated serially and were used to produce quasi-correlograms. The correlograms were plots of the changing values of correlations between two variables through time. These were then examined for peaks and troughs in the various interrelationships.

Moving Cross-Correlations Between Morbidity and Weather

There is no doubt that the response of male and female wheezers to given weather phenomena is different. It is also evident that these meteorotropisms are not constant but change from season to season. The differences between the seasonal correlations are known. What is not known is the process by which this change in relationship from season to season is effected. It is suggested that such changes are conditioned by physiological adaptation/acclimatisation mechanisms to climatic variation over the course of the year. Is this change sudden or gradual? What are the principal meteorological factors involved? To attempt to answer these questions the weather-morbidity correlation has to be viewed as a function of time and has to be computed as a time series over the course of a year.

From the analysis of peak events in the hospital morbidity analyses and from the synoptic analyses it is observed

that, for certain short periods of time, correlation coefficients of large magnitude can be attained but that these are different for discrete time segments. Correlations calculated over larger lengths of time soon reduce in size and significance. This can be explained by the existence of a changing response to a meteorological element or construct. Too long a time period will include days when the response is positive and days when the response is negative. The two effects will cancel and diminish any correlations between the variables.

From the analysis of peak events in hospital morbidity it was seen that about twenty days was the minimum reasonable length of time for which cross-correlations could be calculated. So it was intended to calculate cross-correlations between the morbidity and weather variables *ad seriatim* in twenty-one day segments. This segment length would give ten days either side of a central point in time (the eleventh day) about which the cross-correlation could be said to act. These could be calculated for the entire year and plotted as a time series that represented the changing relationships over time. Discrete twenty-one day segments, however, would have only described nineteen points on an annual scale and, furthermore, could have seriously diminished any covariation that was taking place at the end of one segment and the beginning of another. To overcome this difficulty the cross-correlations were staggered at weekly intervals. This gave fifty-two moving-cross-correlations in a year. This process was illustrated in

figure 4.4.4. Such moving-cross-correlations were calculated for the entire set of morbidity-weather variables. This changed the focus of study from cross-sectional examinations of either peak morbidity or peak meteorology to that of the continuum of their correlation.

Two hundred and seventy-two series of moving-cross-correlations were produced. This number of morbidity-weather permutations was obviously unmanageable and so only the correlograms of the most significant relationships shown by earlier investigations were constructed for comparison and discussion. Because of the results of previous analyses that emphasised the importance of cooling effects, the first selected the variables that measured this dimension; the extremes of temperature, 9 a.m. and 3 p.m. atmospheric cooling power and the 3 p.m. heat stress. The discussions of each of these was organised by geographic region to enhance clarity. Correlograms for pressure and rain were also discussed. Finally, for a change of perspective, Hobart's correlograms for temperature were brought together for comparison.

i) Maximum and Minimum Temperatures

These two measurements are important from two considerations. First, they represent extremes of temperature met with in the course of a twenty-four hour period and therefore indicate the limits of meteorological stress at any given time. Second, they are the most commonly available meteorological measurements. These can usually be obtained

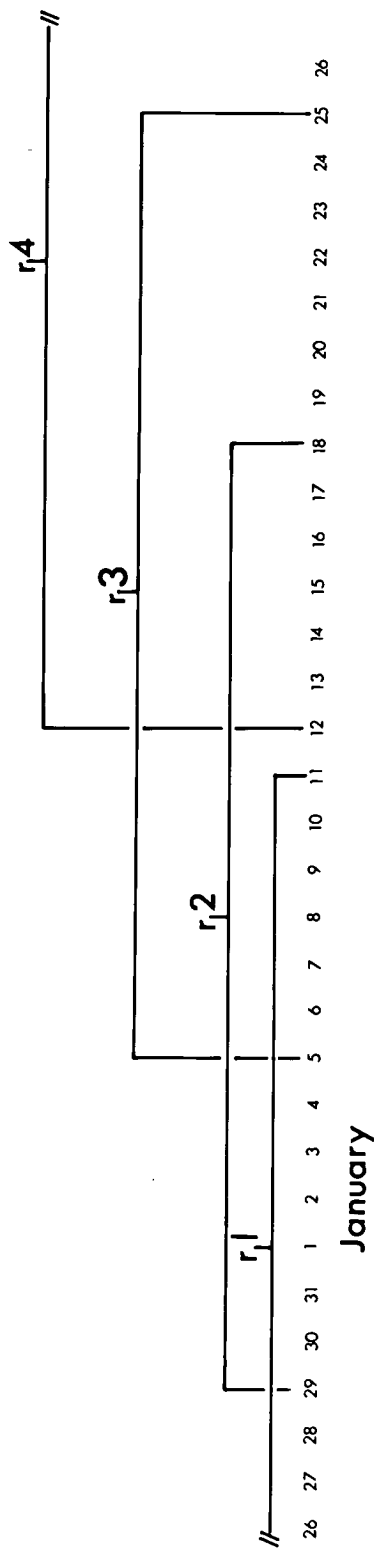


Figure 4.4.4 Moving Correlations: The calculation of 21 day correlations staggered at weekly intervals.

from the majority of the smaller weather stations when other data are unavailable.

Figure 4.4.5 illustrated the serial correlations calculated between the maximum and minimum temperatures and male and female weighted morbidity for four locations. Only weighted morbidity correlations were graphed. This was to avoid unnecessary duplication of figures and description. The correlations were calculated for both raw and weighted data and compared. The results were almost identical. Occasionally the coefficients would differ by as much as, 0.1, but this was rare. Generally the weighted morbidity correlations were less than those with the raw incidence of wheezing. Because of the large amount of data and the desire to obtain readable information after reduction, the entire series of correlations was not plotted. Instead, the middle six months were graphed; from January to June 1978.

From the figure it is evident immediately that the cross-correlations are not constant over time. The coefficients show, even with six months, a wide range of variability. These variable correlations underline the causes of weak associations when coefficients are calculated for periods longer than the fluctuations shown in the graphs. What is also apparent from the charts are the differences shown by the separate sexes and geographic areas. Many of these correlations, however, are attainable purely by chance. To limit discussion mainly to those correlations that are statistically significant, the appropriate

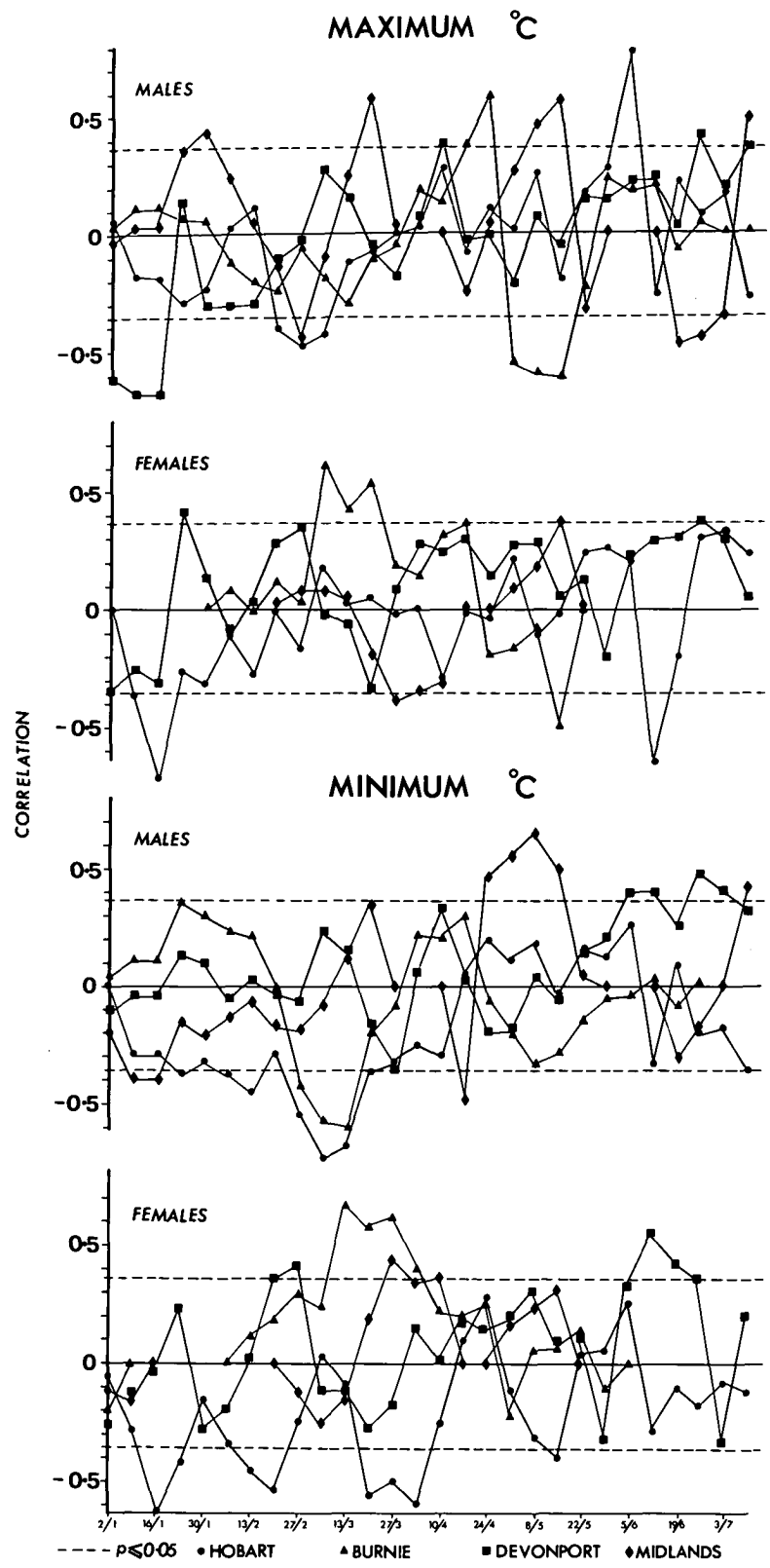


Figure 4.4.5 Moving Cross-Correlations Between Wheezing and Maximum and Minimum Temperatures by Sex and Area.

five per cent confidence limits are drawn on each set of cross-correlations. When $r \geq 0.36$ or ≤ -0.36 there is only a five per cent probability of this level of correlation occurring by chance.

Maximum temperature and male morbidity correlations did not show much agreement from place to place. Exceptions were Hobart and the Midlands which in late February both demonstrated a significant negative correlation. This meant that at this time of the year, 17th February - 19th March, low maximum temperatures were related to high morbidity. This relationship lasted for about four to six weeks. Earlier in January, Devonport morbidity had demonstrated a similar relationship, $r = -0.68$, $p \leq 0.001$. Hobart's Autumn relationship with low maximum temperature was understandable from a consideration of the cooling effect coming at the end of Summer. Devonport's mid-Summer, low-maximum relationship emphasised the importance of any situation that lowered the maximum temperature at this time of the year. Burnie's morbidity produced a similar correlation in May and the Midland's morbidity in June.

Significant, positive correlations between maximum temperatures and masculine morbidity also existed. Midlands males gave a significant peak about every seven or eight weeks at the end of January, in the middle of March, in the middle of May and early in July. The relationship seemed to be cyclical with a period of seven- and one-half weeks and an amplitude of $r = \pm 0.45$. Devonport's morbidity gained significant correlations twice; at the beginning of April and at the end of June. Burnie was a month behind Devonport with its sole peak at the end of

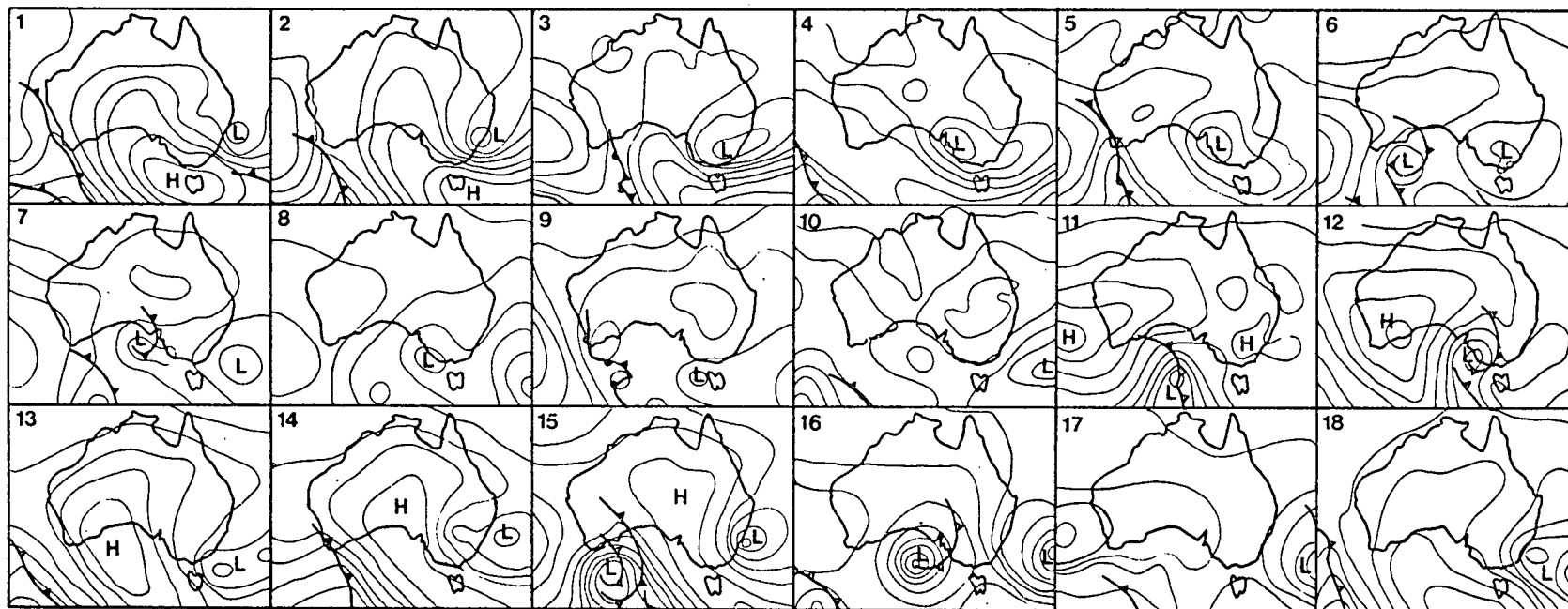


Figure 4.4.6 The Synoptic Progression During Early June 1978.

April. The highest correlation of all was demonstrated by Hobart morbidity at the beginning of June, $r = 0.76$, $p \leq 0.001$. An examination of the synoptic charts for this time showed the extended activity of cyclones off the East coast of Australia alternating with intense cyclones from the Southern Ocean (see figure 4.4.6). The advection of air masses from the far North and South would have generated major weather events and would, in doing so, have accomplished large temperature fluctuations.

The cross-correlations with minimum temperature were just as variable over time but were different to those with maximum temperature. Negative correlations were first demonstrated by the Midlands males in mid-January. This probably reflected the effect of radiation cooling on clear Summer nights in the more continental areas of the state. Midlands males gained a significant negative correlation also in mid-April. Hobart and Burnie showed remarkable parallelism from mid-March. During this Autumn period Hobart's coefficient was -0.72 , $p \leq 0.001$. The relationship was significant at $p \leq 0.05$ for at least six weeks. Positive correlation with minimum temperature was attained during late Autumn in the Midlands. This time, again, was associated with intense cyclone activity advecting maritime air across the state and thus mediating seasonal cooling. Devonport morbidity gained its positive correlations during most of the month of June. This would have been related to maritime air mass advection.

Female morbidity and its correlation with maximum temperature was different to that of males. There did not appear

to be much parallel between the sexes' relationship with this variable. Burnie females gained a negative correlation in mid-May, just as their male counterparts had. Midlands females just gained a significant negative correlation at the end of April. The most outstanding patterns from these series were the two sharp peaks of negative correlations exhibited by the Hobart females. These were in the middle of January and in the middle of June; almost exactly six months apart. The June peak coincided with the trough on the male graph after its high positive peak a week earlier. Whatever caused the high negative peak in the female relationship probably curtailed the high positive peak in the male correlation series. Devonport demonstrated a minor positive peak in late January. The major positive correlation was found in Burnie females during March; a period dominated by anticyclonic stagnation.

The correlations between female morbidity and minimum temperature are dissimilar both to the male and to other female patterns. The only area to show significant negative correlations with minimum temperatures is Hobart. Another cyclical pattern seems to be evident here, with a period of about five-and-one-half weeks. The first negative peak is in mid-January, the same time as the negative peak with maximum temperature. The second is in February, the third in March/April and the fourth in mid-May. Hobart morbidity does not show any positive correlations of any significance. The major positive association is with Burnie's female morbidity from mid March to early April. This peak is a week later and more

extensive than the very similar peak with maximum temperature. Midlands girls gave a significant peak, also during March. Devonport demonstrated two positive peaks, a small one at the end of February and a larger one in mid-June.

If both variables, both sexes and the four areas are considered simultaneously, the data can be seen to describe four basic points. First, the relationships between morbidity and weather are sex and place dependent. Second, the cross-correlations vary both in degree and sign during time. Third, a few morbidity-weather interrelationships appear to demonstrate cyclical behaviour with periods between five and eight weeks. Fourth, in some instances, parallel patterns can be detected between the two sexes' patterns for the same area, or between the extremes of weather for the same sex and area. A cursory examination of synoptic material for one of the peak periods reveals the importance of strongly fluctuating weather systems accompanied by the latitudinal transfer of air masses. The sometimes parallel and sometimes opposite relationships between weather-morbidity correlation peaks could be explained by the dominant characteristics of discrete air masses.

ii) Diurnal Range in Temperature and 9 a.m. Cooling Power

This variable is the difference between the daily minima and maxima (Figure 4.4.7). Negative correlations reflect an association between morbidity and a narrow range in temperature during the day and, positive correlations, a wide range. Devonport and the Midlands were the only areas to exhibit

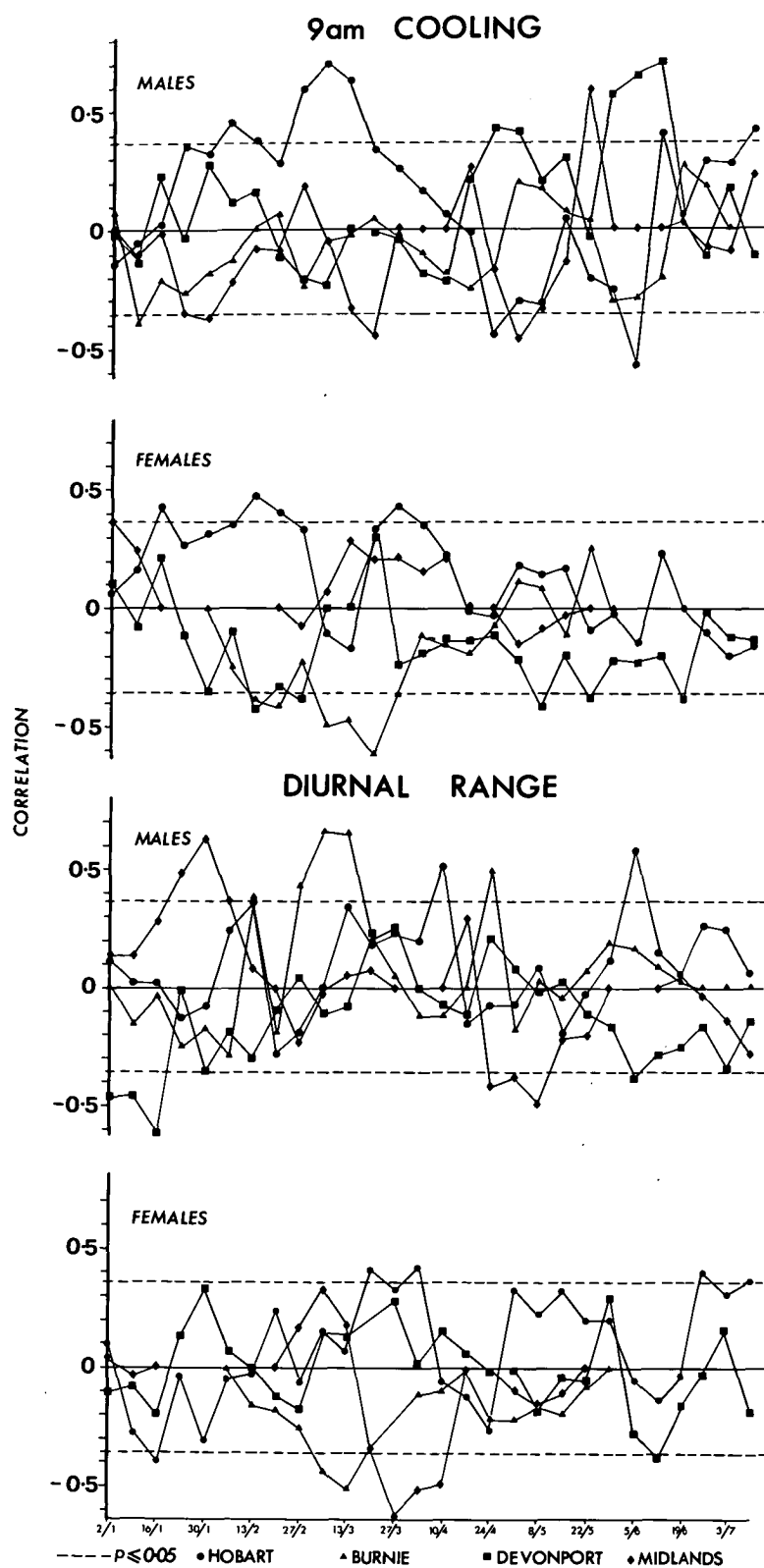


Figure 4.4.7 Moving Cross-Correlations Between Wheezing and 9 am Cooling Power and Diurnal Range in Temperature by Sex and Area.

masculine negative correlations with diurnal range. Devonport's correlations are in the middle of Summer (16th January) and the middle of Winter (5th June). In the Midlands a significant negative correlation is found at the end of April and the beginning of May. Positive correlations in the Midlands males are found at the end of January. For Burnie, these occur in March and in April, for Hobart in the middle of April and at the beginning of June.

Far fewer significant correlations were discovered with female morbidity. Hobart and Devonport barely made significant negative correlations in January and June respectively. Burnie showed significant association in early March and the Midlands in late March-early April. The only positive correlations were with Hobart female morbidity. This was observed in mid-March and late June.

The other variable illustrated in figure 4.4.7 was the 9 a.m. cooling power. Negative masculine correlations with this index were of minor significance until Hobart's peak in early June. Smaller peaks were for Burnie in January, mid-March and late April-early May. The positive correlations with 9a.m. cooling were generally more significant. The Midlands had a strong peak at the end of May. This was followed by a more extended peak in Devonport and was preceded by a smaller Devonport peak at the end of April. Hobart morbidity had the most extended positive association with cooling, from the end of February until late March.

Females during this time did not exhibit as significant correlations as the males. Negative coefficients were found with both Burnie and Devonport data. Burnie's were during late February and March. Devonport had smaller correlations at this time and also later during May and June. The only positive correlations discovered were with Hobart morbidity in January, February and March.

iii) 3 p.m. Heating and Cooling

The 3 p.m. cooling generally possessed low correlation coefficients with morbidity (see figure 4.4.8). This was linked to the fact that 3 p.m. is close to the usual time of daily maximum temperature and minimum cooling. Negative correlations between Midlands male morbidity and afternoon cooling were found at the end of January and the beginning of June. For Devonport males a negative correlation in mid-January was followed by a positive correlation at the end of that month. Hobart and the Midlands gave significant positive correlations at this time. The largest correlation was found between Devonport and 3 p.m. cooling during early June, $r = 0.47$, $p \leq 0.05$. The female patterns were similarly subdued. Burnie, at the end of February, possessed a negative correlation and at the end of May, a positive one. Hobart demonstrated a positive correlation in mid-June.

Heating was more appropriate to the time of day and gained bigger correlations. Hobart male morbidity gave a negative correlation with heat-stress in Summer and a positive

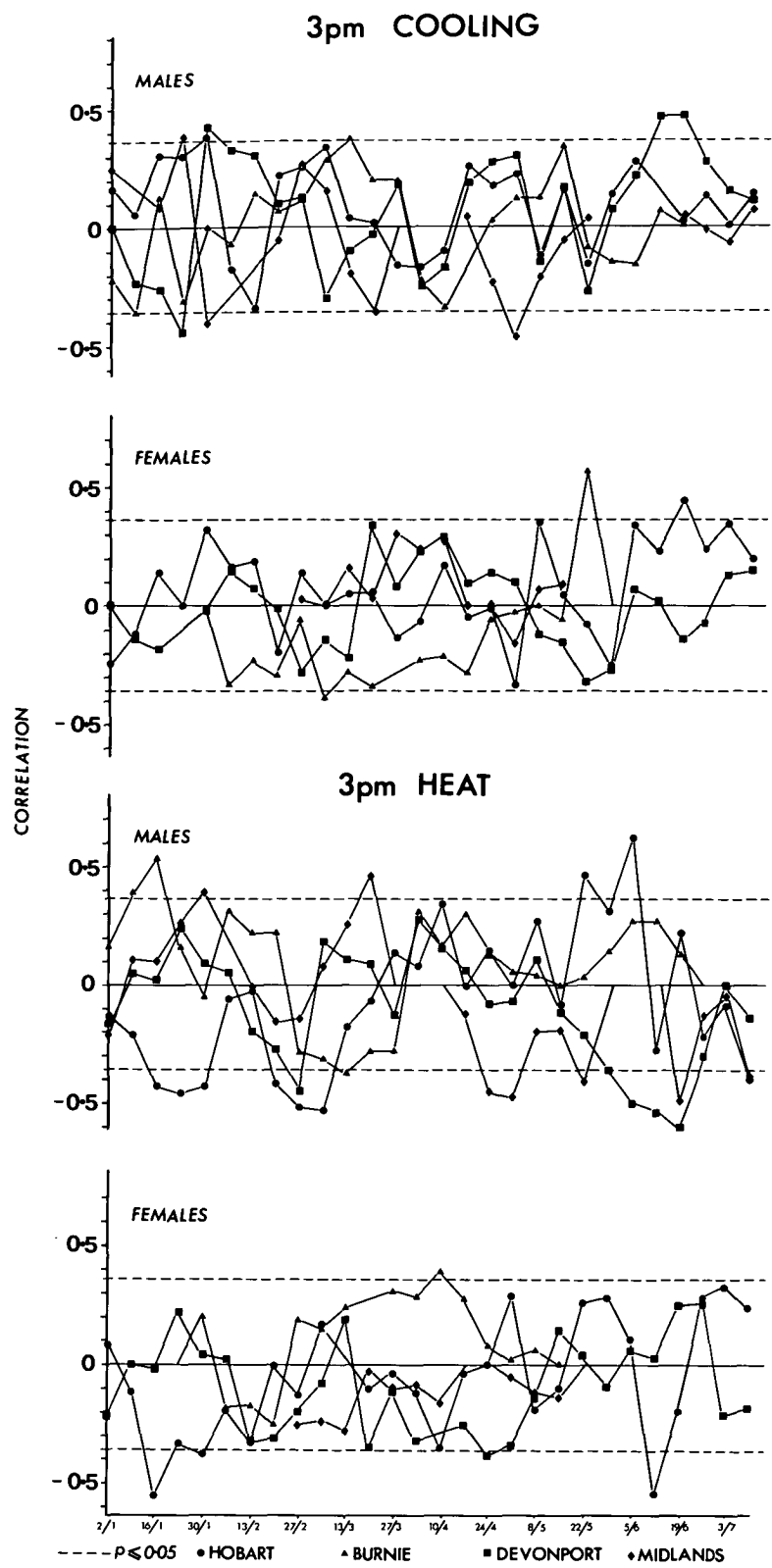


Figure 4.4.8 Moving Cross-Correlations Between Wheezing and 3pm Cooling Power and 3pm Heat Stress by Sex and Area.

one in Winter. Other negative correlations were shown by Devonport males at the end of February and during June; and by the Midlands in late April and late May. Positive correlations were given by Burnie in January, the Midlands in March and Hobart in May-June. In females the only positive significant correlation was for Burnie, in April. Hobart females gave two negative peaks, one in mid-January and one in early June. These were very close to the correlations given in figure 4.3.3 for temperature.

iv) Pressure and Rain

The series of correlations for these two weather elements are given in figure 4.4.9. Pressure can be related to cyclonic or anticyclonic dominance by negative and positive correlation respectively. Significant negative peak correlation periods occur as early as February in Devonport male morbidity. Shortly afterwards in early March the Midlands and Burnie males share a negative peak. Hobart male morbidity does not demonstrate a significant negative association with 3 p.m. pressure until June. It has a small peak at the beginning of this month and a large one at the end, $r = -0.58$, $p \leq 0.01$.

A cluster of significant positive correlations occur in January and February associated with Summer anti-cyclones. Burnie males produce peaks in each of these months and Devonport shows a minor peak in mid-January and another in early-May. Co-occurring with the second Burnie peak in February is a peak for Hobart male morbidity. Right at the end of February comes

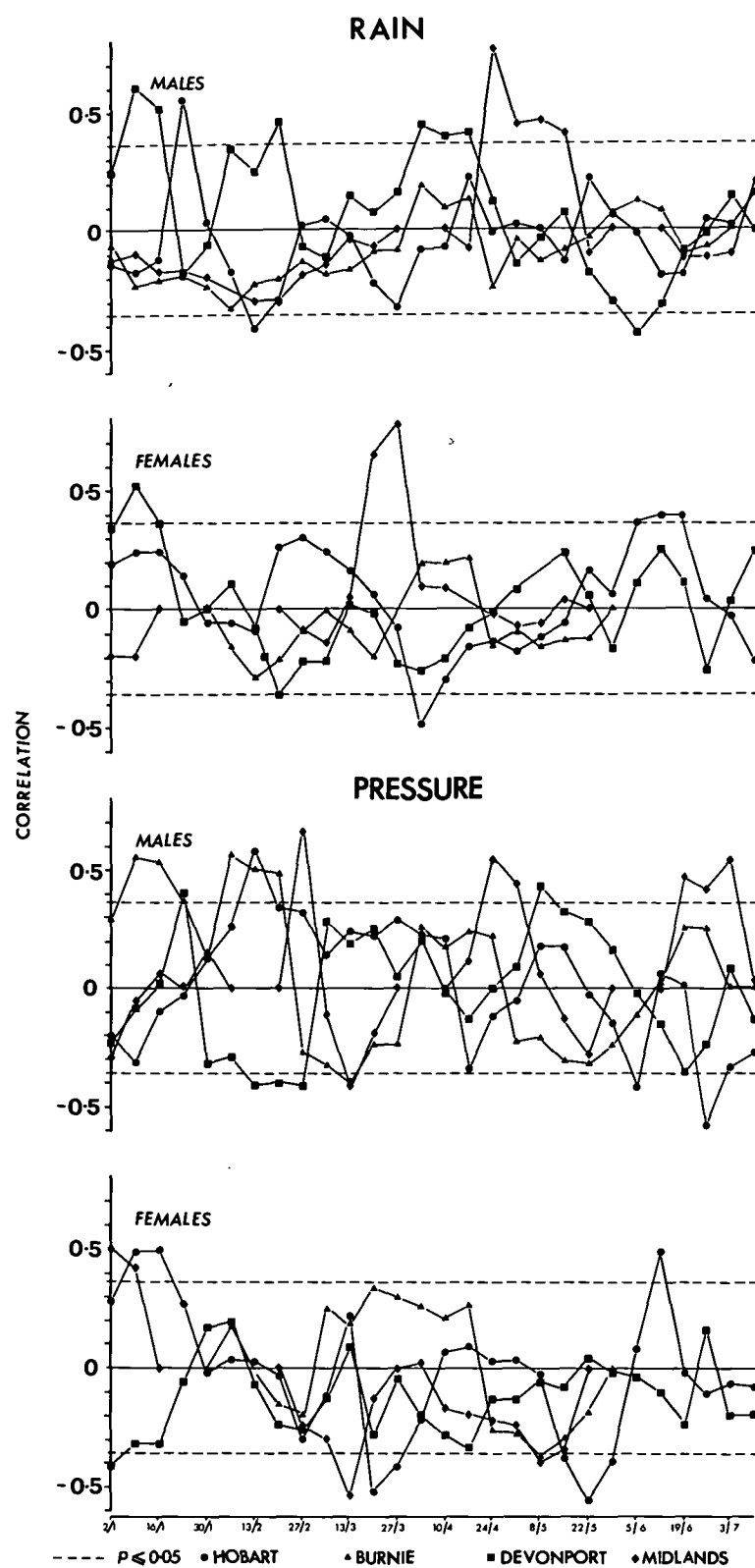


Figure 4.4.9 Moving Cross-Correlations Between Wheezing and Rain and Pressure by Sex and Area.

the maximum Midlands correlation, $r = 0.63$, $p \leq 0.001$. The Midlands males actually exhibit three peaks, each about eight weeks apart.

Female morbidity, once more, illustrates an entirely different set of relationships. Devonport morbidity shows one significant negative correlation in early January and then reduces to insignificance. The Midlands correlations are negative in mid-March and in early May. Hobart shows two strong negative peaks in late March and late May. These two Hobart Autumn peaks are balanced by positive peaks in Summer and Winter. Only one other positive correlation is found in female morbidity, that is with Midlands females in January.

Correlations with rainfall seem to be more parallel between the different places and closer to zero on average. Male negative correlations with rainfall occur twice, once with Hobart morbidity in February and once with Devonport morbidity in June. The positive correlations were larger and more numerous. Devonport male morbidity demonstrated three peak periods, January, February and April. Hobart's peak occurred two weeks after Devonport's early January peak. The outstanding period of correlation with rainfall, however was shown by Midland males' morbidity in late April-early May. The correlation coefficient peaked at 0.76 , $p \leq 0.001$ and then reduced in size but maintained significance for another month.

For female morbidity only one negative correlation with rainfall was found. This was Hobart's morbidity at the beginning of April. Hobart also gained a significant positive association

in early June. Two other positive correlations were important. The first was with Devonport morbidity in early January. The second was the Midland females' morbidity in March. Like the Midlands males' peak, this was the most significant positive correlation shown. It peaked on the 27th of March at $r = 0.78$, $p \leq 0.001$ after gaining a correlation in the previous week of $r = 0.65$, $p \leq 0.001$.

v) Hobart

To conclude this discussion of serial correlation and to illustrate some interesting phenomena that are not easily observed from the figures of six months' morbidity, a year's moving correlations are given for Hobart from July 1977 to June 1978. Two graphs are given, one is for maximum and minimum temperatures and the other is for 3 p.m. cooling power and heat stress (see figure 4.4.10). The correlations used to construct these graphs are those obtained from the incidence of wheezing rather than those of weighted severity. This is for two reasons. First, the differences between the two indices are very small. Second, the last six months of these figures enable comparisons to be made with Hobart morbidity graphs in previous figures in order to examine these differences.

The impression of variation in relationships that is gained from the previous figures in this section is reinforced by viewing a whole year at once. The variability does not seem to be constant or regular from the graphs of Hobart's morbidity's correlations. Strong fluctuations are observed in

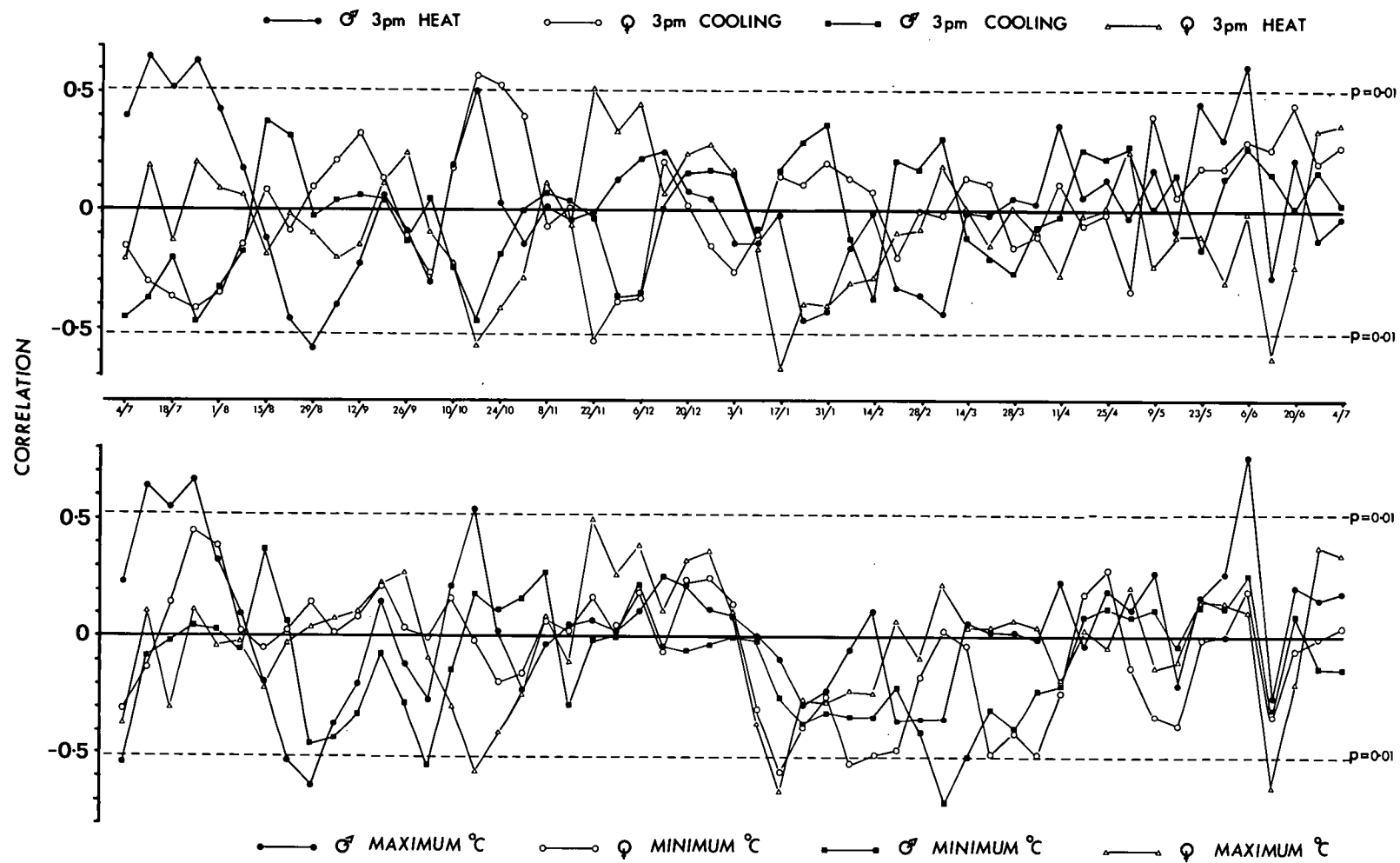


Figure 4.4.10 Moving Cross-Correlations Between Wheezing and Atmospheric Parameters in Hobart, July 1977 - July 1978.

the Winter and Spring months. These flatten during Summer and early Autumn and then are seen to start fluctuating again in early Winter.

Certain series appear to agree closely, others only for isolated periods. For example, morbidity correlations with maximum temperature and 3 p.m. heat-stress are almost identical for the separate sexes. The identical correlations for the different sexes, however, are not the same. The female morbidity correlations with maximum temperature are not usually parallel with the male morbidity correlations and are often opposite. A good example of this is the correlation for the 17th of October, 1977. Here, females have a negative correlation with maximum temperature, $r = -0.58$, $p \leq 0.01$, and males have a positive correlation, $r = 0.54$, $p \leq 0.01$. For either sex, the maximum and minimum temperature morbidity correlations usually agree or at least do not disagree significantly. Close association is observed between female maximum and minimum temperature correlations during most of November, December and January. An event during the 6th to the 22nd of June, 1978 causes all of the male and female maxima, minima and 3 p.m. heat-stress correlations with morbidity to co-vary in a sudden, strong, negative dip.

The sexes' opposite correlations are nowhere better exemplified than in the graph of correlations with 3 p.m. heating and cooling. The male morbidity responses to heat is usually the opposite to that of cold and the opposite to that of female morbidity. From this it follows that the male morbidity

correlation with cooling should be similar to the female morbidity correlation with heat stress and *vice versa*. This pattern is followed for about half of the time. The relationships between the male and female correlation series actually seem to alternate from being opposite to each other to being the same. This is only discerned when the complete spectrum of coefficients is taken into account. It is not easily detected from an examination of the peaks. The best example is given by the correlations between the months of October and December. By the 17th of October male morbidity's correlation with heat-stress was 0.51 compared to the females coefficient of -0.57. The correlations for cooling power for the same period are -0.46 and 0.58 respectively. This describes a situation where the sexes' response was opposite. Two months later, by the 6th of December the male morbidity's correlation with heat-stress was 0.23 and the female equivalent was 0.46. The correlation for cooling for the same period are -0.36 and -0.37 respectively. The situation has changed to one where the sexes maintain the same relationship with these weather variables. All of the coefficients above are significant at $p \leq 0.05$ with the exception of $r = 0.23$.

For the six months' of data analysed before, five per cent confidence interval were drawn in order to indicate the significant peak events. With a year's data the one per cent levels were chosen to reduce the number of events for discussion. This centred upon three events which were likely to have occurred only one time in one-hundred due to chance.

In July 1977, the major cross-correlations were between male morbidity and the maximum temperature and 3 p.m. heat-stress where $r = 0.66$, $p \leq 0.001$. This month's weather was dominated by a succession of anticyclones which advected warm continental air from the Australian mainland over Tasmania following a series of extremely stormy Westerlies. On the 30th of June the maximum temperature had been only 6.36° Celsius, by the 7th of July it was 15.2° . This rapid change in thermal stress may have upset the population's morbidity limit. Interestingly, at the start of the year the male morbidity - minimum temperature correlation was highly negative, $r = -0.54$, $p \leq 0.001$. On this occasion also, male and female morbidity seemed to co-vary. On the 25th of July when the male correlation with maximum temperature was 0.66 , $p \leq 0.001$ the female correlation was 0.45 , $p \leq 0.05$.

On the 29th of August the male morbidity correlations with maximum temperature and with 3 p.m. heat-stress were negative, $r = -0.62$ and -0.58 , $p \leq 0.01$. The female correlations are associated with a series of cold fronts and stormy Westerlies that occurred at this time. These weather events would have produced a period of strong atmospheric cooling. Also, during this time male morbidity and minimum temperature were correlated $r = -0.46$, $p \leq 0.05$.

The next major event occurred during mid-October. Males and females demonstrated opposite relationships between their morbidity and the thermal environment. Males correlated positively with 3 p.m. heat-stress and maximum temperature,

$r = 0.52$ and 0.54 , $p \leq 0.01$ and negatively with 3 p.m. cooling, $r = -0.46$, $p \leq 0.05$. Females, conversely, correlated positively with 3 p.m. cooling, $r = 0.58$, $p \leq 0.01$, and negatively with 3 p.m. heat-stress and maximum temperature, $r = -0.56$ and -0.58 , $p \leq 0.01$. This was a period of strongly alternating weather systems. Here, females' morbidity probably related to the westerly and cyclonic components and males' morbidity to the anticyclonic pattern.

On the 22nd of November female morbidity showed a positive correlation with 3 p.m. heat stress and a negative one with 3 p.m. cooling. This was a change from five weeks before. It was linked to the North-South advection of warm humid air by the action of strong anticyclones and a "tropical dip" cyclone towards the end of the month.

During January 1978, the female morbidity correlations changed once more. The maximum and minimum temperature and the 3 p.m. heat-stress all gave negative correlations with female morbidity, $r = -0.66$, -0.58 and -0.66 respectively, $p \leq 0.005$. The male morbidity correlation was also negative, $r = -0.46$, $p \leq 0.05$. In a population adapted to the warmth of Summer, the cold fronts on the 10th, 16th, 21st and 26th probably obtained a stronger meteorotropic response than usual.

In March the correlation between male morbidity and minimum temperature became significantly negative. This correlation was interesting because of the seasonal peaking of male acute episodes observed in this month from other data, for example hospital morbidity. Out of the twenty-one days

used to calculate the coefficient for the 7th of March, on fifteen of these days a cold front was over Tasmania and ten could be described as stormy Westerlies. The "cooling effect" of these systems coming at the end of Summer is self-evident.

Perhaps the most remarkable event in the entire series happened in June 1978. Within the course of two weeks highly positive male morbidity correlations with maximum temperature and 3 p.m. heat-stress fell to zero, and the same coefficient for female morbidity rose from zero to highly negative. On June 6th, the male coefficients were 0.62 with 3 p.m. heat-stress and 0.74 with maximum temperature, $p \leq 0.001$. By the 13th of June they were -0.28 and -0.26 respectively. On the 6th the female correlations were -0.01 with 3 p.m. heat-stress and 0.11 with maximum temperature. On the 13th they were -0.62 and -0.64, $p \leq 0.001$. First male morbidity was strongly positively associated with thermal stress and then a week later, female morbidity was highly negatively associated with the same element.

Figure 4.4.11 shows the synoptic conditions that existed throughout this time. The early parts of the month that gave the positive correlation between male morbidity and thermal stress, were influenced by the juxtaposition of a cyclone off the East Coast of the Australian Continent and an anticyclone over Tasmania. From the 2nd to the 6th the State was perpetually overcast with high humidities and rainfall. This was due to the advection by the synoptic systems of warmer, more humid air from the East. These conditions were related to increased male morbidity. This was followed a week

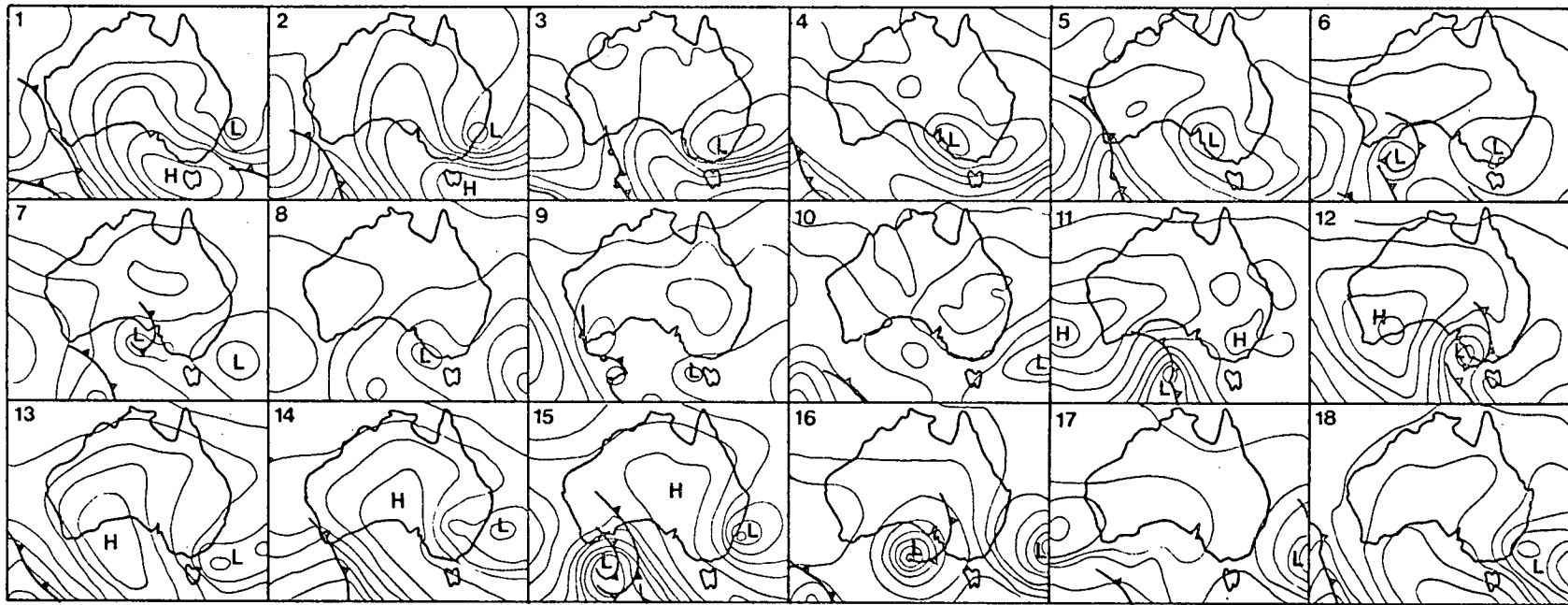


Figure 4.4.11 The Synoptic Progression During Early June 1978.

later by a series of southerly cyclones that brought cold air from the southern trough. Over the 15th to the 17th of June a southerly cyclone to the West of Tasmania converged upon a subtropical cyclone to the East of Tasmania and produced strong weather conditions. These patterns were associated with the increase in female morbidity.

Summary

Correlation analysis has proved to be useful in the detection of morbidity-weather relationships. Initial analysis upon annual or seasonal data demonstrated increases in coefficients with decreases in the sampling period. Later, the definition of weather types and an analysis structured upon their relative frequency illustrated the importance of the synoptic situation. Although at different times of year the cross-correlations were different for a given synoptic situation, these could be explained on a physical basis. Some patterns regularly produced strong relationships, others none.

The most revealing technique used was the method of moving-cross-correlation. Results from this procedure demonstrated the inadequacies of calculating correlations between weather and morbidity over long periods of time. The relationships between many weather and morbidity indices were shown to vary with time and place and sex. Within the course of a single month a highly positive correlation might be seen to become a highly negative one. Some correlations over time were seen to

describe almost cyclical rhythms between high and low peaks. The period of these events was of the order of 6-8 weeks; much less than a season's length. The extreme events were examined in relation to synoptic charts for the same period and strongly fluctuating weather patterns were seen to be associated with these times. The relationship between morbidity and weather was seen to be not a fixed one but a variable one that was dependent upon previous conditions and was modified by strong weather patterns.

4.5 Spectral Analysis of Weather and Asthma Morbidity Relationships

Introduction

Childhood asthma episodes in Tasmania have been shown to exhibit meteorotropisms. The meteorotropic relationships portrayed, however, differed from sex to sex and from place to place. Furthermore, within any one sex and location specific group a given meteorotropism changed over the course of time. In some seasons the cross-correlations were positive; in others negative. For short periods the correlations approached unity, at other times the coefficients were close to zero. A few of the moving cross-correlations actually seemed to exhibit some form of cyclical pattern in their time series. From an examination of several correlation analyses the basic meteorotropic relationship appeared to be linked to thermal stress. Actually, males and females and the different geographic samples demonstrated similar correlation constellations but on different occasions. Therefore, the initial impression of difference in meteorotropic behaviour between the several groups was accentuated by the different temporal patterns in the cross-correlations.

The concept of "morbidity limit" has already been discussed. The idea of acquired resistance to a meteorologically determined insult such as thermal stress has been expressed as a variable threshold that can be modified by environmental stimuli. For example, a recently-stressed asthmatic would not have responded as readily to a repeated stimulus, because his threshold would have been elevated. Similarly, an asthmatic

free of thermal stress for some time would have responded strongly to a cold-shock because the threshold would have been lowered. During the course of a year seasonal influences may have affected this threshold because of the relative occurrence of thermal stress at different times of year. A broad seasonal pattern of adaptation or acclimatisation to the thermal regime would have been expected to exist. This, in part, would have explained the lowered incidence of asthma at the height of Summer and Winter, when the thermal environment was most homogenous, and the heightened incidence in Spring and Autumn, when temperatures were fluctuating most strongly. Seasonal differences between the sexes may have been due to physiological factors that operated differently in boys and girls. Changes in pattern from place to place were obviously related to the change in climatic regime.

Built upon this basic pattern of seasonal threshold variability are the short-term shocks due to intense weather systems. Strong weather at any time of the year brings asthmatic reactions. The reaction to a particular weather event in a warm-adapted population, however, is different to that in a cold-adapted group. A strong cold front occurring in Winter will not have as great an effect as a similar one in the middle of Summer. Meteorotropic response has thus been shown to operate at two scales; that of long-term annual/seasonal fluctuations and that of short-term fluctuations of the order of days or weeks. Sometimes these two levels are positively correlated and at other times they are negatively correlated.

They result in the complex series of interrelationships shown in Section 4.4.4. One approach to unravelling this web of inter-correlations over time is to move from the domain of time to the domain of frequency. It is possible, for example, that asthma attacks could be positively related to long-term meteorological cycles and negatively related to short-term cycles. As the basic concept of frequency analysis is the frequency spectrum, it is commonly termed spectral analysis.

Spectral Analysis : An Introduction

Spectral analysis is based upon the fact that a stationary time series, that is one that does not contain trend and that possesses constant variance, can be decomposed into a theoretically infinite number of cyclical components of different frequencies. Each frequency contributes a proportion of variance to the total variance density spectrum. This spectrum is constructed with the percentage of variance measured on the vertical ordinate and the frequencies in cycles per unit time along the horizontal ordinate. Peaks in the spectrum usually represent dominant waveforms at those frequencies. A flat spectrum is found with random data and is called "white noise". A concentration of variance in the low frequencies is termed "red noise". This is characterised by long waveforms and trend. A large amount of variance at high frequencies is known as "blue noise". The smallest of these short wavelengths is twice the length of the basic measurement interval. In this

study the interval was one day and the highest resolution was therefore, 0.5 cycles per day or one cycle in two days.

Variations such as diurnal rhythms are undetected by this method. Similarly because of the short time period of the study, very long wave forms, (≥ 8 weeks) were not well-resolved. Usually the length of data needs to be at least five times the length of the maximum waveform of interest. To establish the presence of an annual cycle one would need at least five years of data.

Geographer's interest in spectral analysis has been linked to the growth of digital computers and the availability of the Fast Fourier Transform (FFT). Rayner,²⁶ reviewed the historical progress of the methodology in his excellent introduction to the subject. Much recent work in this area has been produced by students of the Bristol school of geography.²⁷ Earlier reviews of technique were supplied by Panofsky,²⁸ and Munn.²⁹

The Fourier transform can be thought of in much the same way as a mathematical operator. Its function is to transform time-based information into frequency information. Modern spectral methods involve the Fourier transform of the autocovariance function (ACF) to obtain the variance spectrum; it describes the amount of variation for each unit of frequency. Cross-spectral analysis is merely the extension of spectral methods to the simultaneous treatment of two variables.

Cross-spectral analysis is used in the exploration of time series for lagged relationships. Its principal aim is to derive hypotheses and parameters upon which orthodox least-squares models can be built. Lagged relationships that are difficult

to detect in the time domain because of perturbations in sequence have their variance attributed to the correct frequency bands in the spectra. The cross-spectra therefore, describe lagged relationships more perfectly than temporal cross-correlations.

Pairs of weather and morbidity elements are taken in the following analysis and subjected to cross-spectral analysis. The computer sub-routines used are from I.M.S.L.³⁰ Subroutines, FTCROS and FTFREQ produce as output, the individual series' means, variances and autocovariances; the cross-covariances, individual spectra and the cross-spectrum estimates. The cross-spectrum is made up of two additive parts, the co-spectrum and the quadrature spectrum. The co-spectrum measures the in-phase components of the cross-spectrum amplitudes and the quadrature spectrum measures the out-of-phase components. Two other spectra are also produced, the phase spectrum and the coherence spectrum. Phase is calculated for each frequency and describes the amount by which the frequency bands are lagged in fractions of a wavelength. Wavelength is, of course, the reciprocal of frequency. Coherence is an estimate of the correlation between each pair of frequency bands. Phase and coherence, together, completely describe any bivariate, normal stochastic process.

Autocorrelations and Cross-correlations

Before the discussion starts upon frequency material it is worthwhile to inspect the preliminary output from the spectral analysis that describes variation in the time domain. These are

the auto-correlation and cross-correlation coefficients. Both can be plotted as correlograms where the vertical ordinate is from -1 to +1 and the horizontal axis is a time scale that measures units of lag.

Because of the non-independent nature of neighbouring values in a time series, the significance of the individual correlations cannot be estimated. The correlograms in figure 4.5.1 have to be interpreted solely on the basis of their shape. When there exists positive auto-correlation the correlations decay smoothly and exponentially whereas negative auto-correlations produce an oscillatory and exponential decay of successive correlations. In a time series that contains persistence but not trend the auto-correlation coefficient becomes very small given a sufficiently large time-lag. Where trend exists, the auto-correlation function levels off parallel to the horizontal ordinate. If the series contains parts of low frequency cycles the function is much extended before it eventually reaches zero.

The correlograms for the sexes morbidity in the four areas are given in figure 4.5.1. For the first few days the male and female graphs, for any area, follow each other quite closely and then diverge to describe different relationships. The best agreement between the sexes' correlograms is seen in the Midlands and the most disagreement is seen in the graphs for Hobart. The shortest persistence is observed in the Devonport correlograms where the auto-correlations decay to zero in three or four days. The longest persistence is attained by Burnie boys and Hobart girls; their auto-correlations reach zero in fifty and forty days lag respectively.

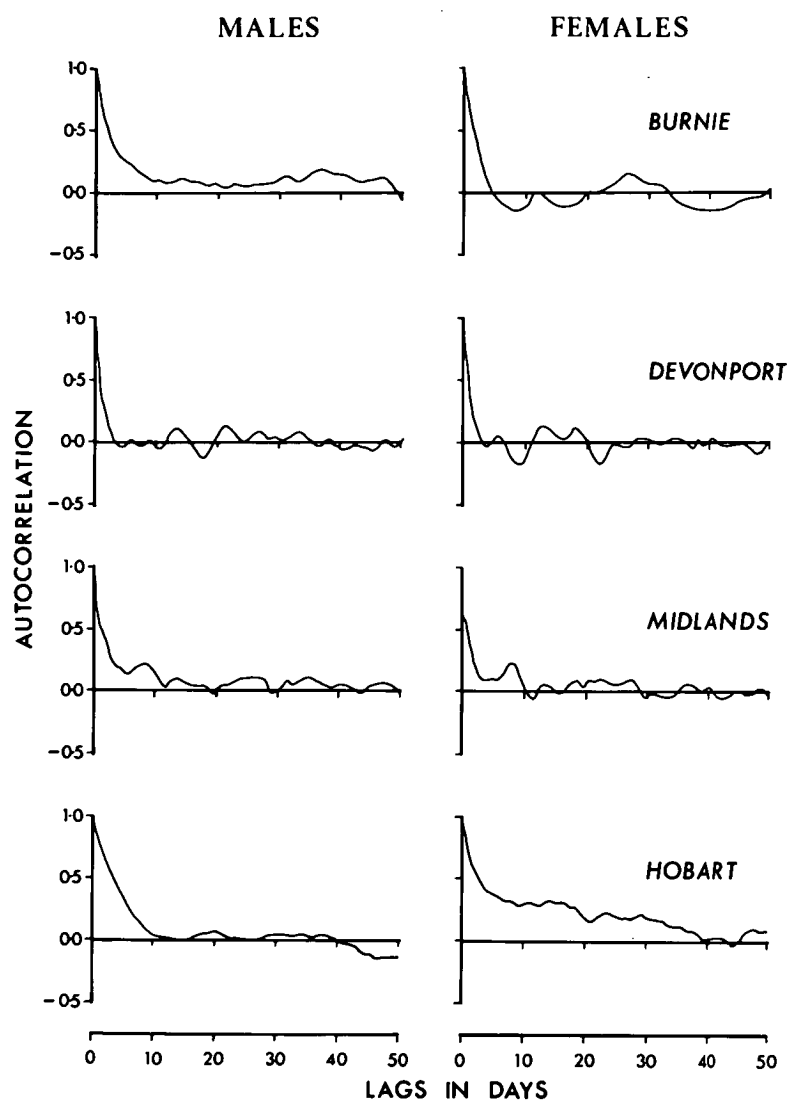


Figure 4.5.1 Auto-correlograms of the Wheezing Series for Each Area and Sex.

Each correlogram, although unique, most closely resembles the other correlogram from the same area. This is particularly obvious with the Midlands and Devonport examples but is more difficult to observe in the Hobart and Burnie figures. In the Midlands, both sexes dampen quickly with the first four lags and then exhibit a minor peak around eight and nine days lag. In Devonport this quick dampening is also apparent; both sexes' correlograms reach zero in three or four days lag. They then oscillate about zero in an opposite fashion. The steep drop in auto-correlation seen in these graphs indicates a lack of long periodicities in the data. The morbidity in these locations is of a short-term nature with little persistence or seasonality. Peaks in the correlograms may represent cyclical patterns present in the morbidity series.

In Hobart and Burnie the dampening is not so steep. Burnie females' auto-correlogram is the most similar to Devonport and Hobart. It dampens to zero in five days lag and then oscillates with a period of about thirty days. Burnie males' correlogram, however, flattens out at about 0.1, after nine days lag and does not reach zero until lag fifty. This pattern indicates the presence of trend or long-term cycles in the morbidity. This is also found in the Hobart graphs, particularly the female one. Hobart females' correlogram after fifteen days maintains a coefficient of 0.3 and does not reach zero until the fortieth lag. Hobart males' correlogram differs in that it decreases more quickly. By the tenth day of lag it is practically zero.

Two locations are seen to be dominated by short-term morbidity events and two by long-term (seasonal?) cycles and trend. Why Burnie and Devonport should fall into opposite types demands thought and investigation. Hobart and Burnie are the most urbanised and industrialised sites and both demonstrate the long-term effects. The Midlands is, of course, rural and Devonport is generally held to be a much cleaner city than Burnie, its near neighbour on the North West Coast.

The cross-correlogram is the bivariate extension of this technique. Figures 4.5.2 to 4.5.6 illustrate examples of this type of correlogram for four pairs each from Burnie, Devonport and the Midlands. Cross-correlograms of weighted morbidity and both maximum and minimum temperatures are given for each area and in addition, 9 a.m. cooling power and 3 p.m. cooling power correlograms are given for Hobart.

Hobart

In Hobart, both sexes start with negative correlations with both maximum and minimum temperatures (figure 4.5.2). Females possess the largest coefficients in both cases. Although different in absolute terms, both correlograms show parallel peaks and troughs and trend. The two graphs are very close at first and then part after about two weeks of lag. The male correlogram with maximum temperatures reaches zero after seventy-five days lag and generally remains positive after that time. The female correlogram does not meet zero in 150 days of lag. Minimum temperature correlograms are a little different. First,

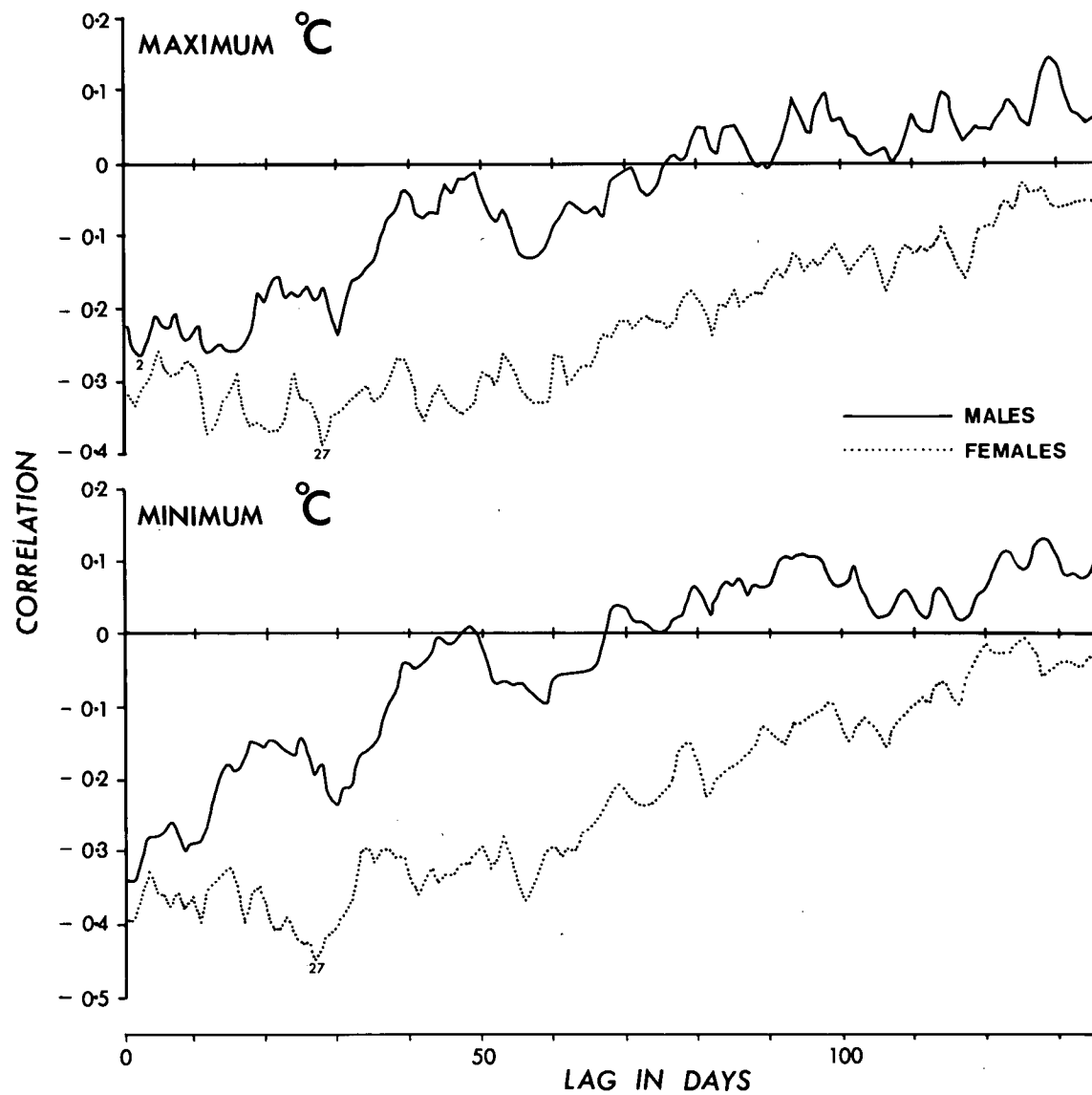


Figure 4.5.2 Cross-Correlograms Between Maximum and Minimum Temperatures and Wheezing in Hobart.

their origin is of greater magnitude and second, their variability seems to be greater. The male correlogram crosses zero at forty-seven days lag and then returns to negative before becoming positive on the sixty-seventh lag. The female correlogram never reaches zero in 150 lags.

In the male correlogram with maximum temperature the coefficient at zero lag is -0.23 ; this peaks at two days lag, $r = -0.27$. For females the coefficients are -0.32 at zero lag and -0.39 at a twenty-seven day lag. With minimum temperatures the male correlation at zero lag is the highest, $r = -0.34$. Females correlation at zero lag is -0.39 , this increases to -0.45 at twenty-seven days lag. In both the maximum and minimum correlograms males demonstrate subsidiary peaks at twenty-nine days lag.

In the correlograms with morning and afternoon cooling power the origins become positive with the female graph possessing the greatest magnitude (figure 4.5.3). A comparison of the correlograms for morning (9 a.m.) cooling and minimum temperature reveals almost mirror-image relationships. The coefficients are about the same magnitude but their sign is reversed and the same fluctuations occur in both sets of graphs. The male correlogram with 9 a.m. cooling power at zero lag, $r = 0.35$, has a minor peak at twenty-nine days lag, $r = 0.29$, and reaches zero at sixty-eight days lag. At lag zero, the female coefficient is 0.39 , this reaches 0.43 at twenty-seven days lag and never drops to zero in 150 days.

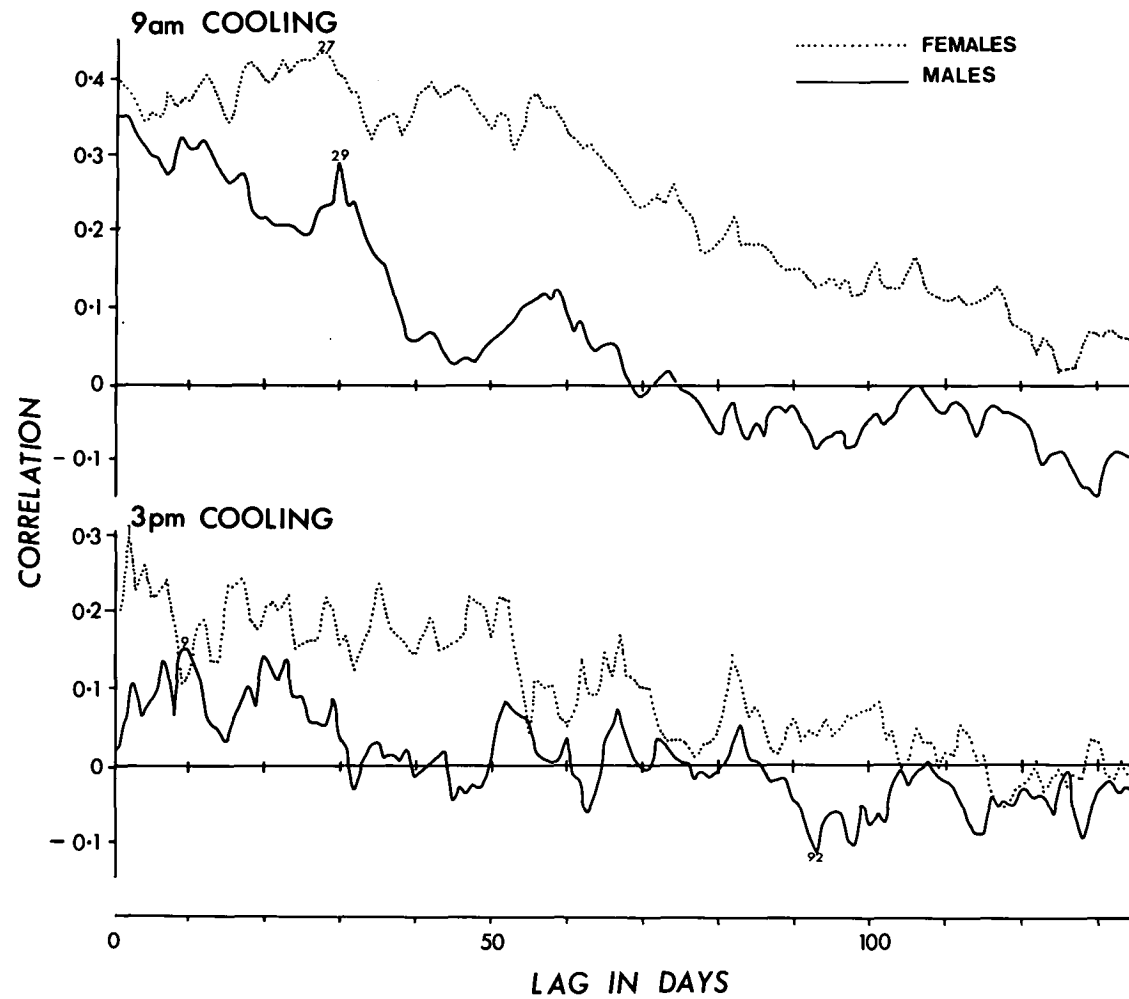


Figure 4.5.3 Cross-Correlograms Between 9am and 3pm Cooling Power and Wheezing in Hobart.

The two sexes show the same basic pattern as the morning cooling but the range between them is much narrower and the graphs are more spiky. At zero lag the female and male coefficients are 0.2 and 0.02 respectively. The female correlogram peaks with a one day lag at $r = 0.29$. The males' highest correlation is 0.15 at nine days lag. Reaching zero at thirty-one days lag, the male correlogram then proceeds to oscillate about zero. It gains its highest negative correlation at ninety-two days lag, $r = -0.12$. The female graph reaches zero at one hundred and four days lag. Interpretation of these results will be discussed with other areas' results at the end of this sub-section.

Midlands

The Midlands correlograms present a different picture to those for Hobart (figure 4.5.4). The decrease in magnitude of the coefficients may be linked to the smaller number of asthmatics in the sample. Any interpretation should proceed with caution. The male correlation with maximum temperature at zero lag is -0.02 this changes to 0.02 at three days lag and by nine days lag peaks at -0.11. The maximum peak is at eighty-four days lag, $r = -0.13$. Females' correlations start at zero lag with $r = -0.18$ and peaks at twenty-eight days lag at -0.22. The female correlogram with maximum temperature eventually reaches zero at one hundred and four days lag and then oscillates weakly. Although the male correlogram zeroes at three, forty-seven, and fifty-one days lag it remains negative

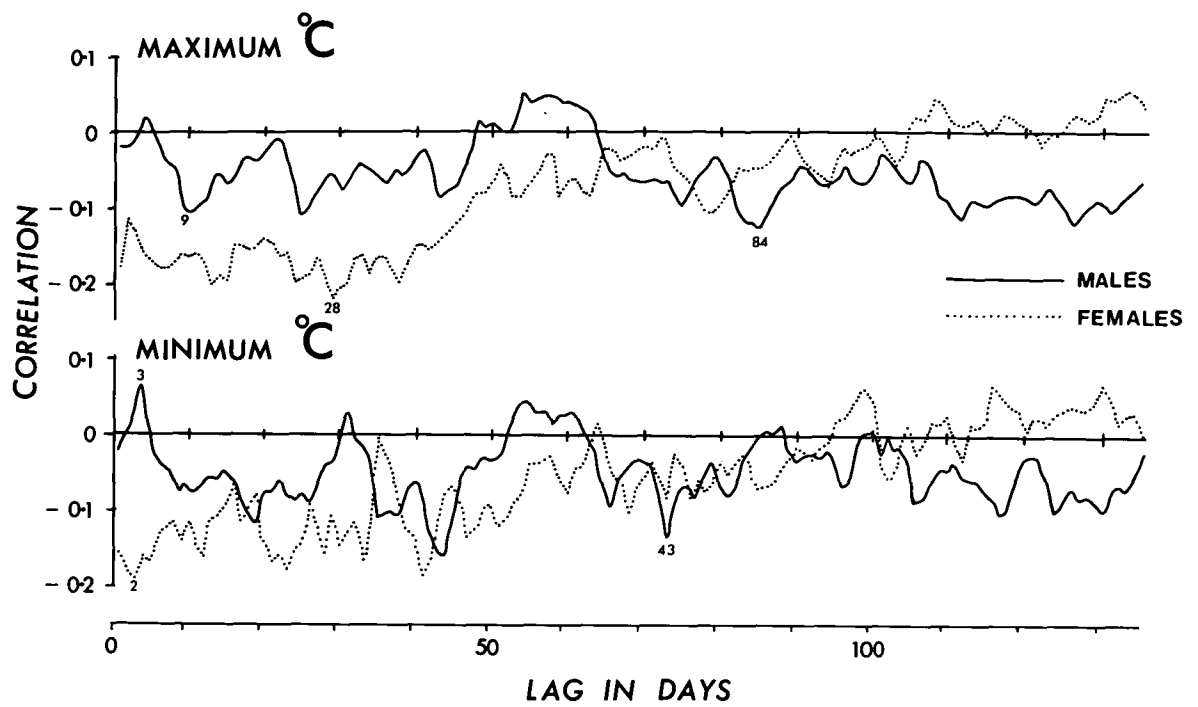


Figure 4.5.4 Cross-Correlograms Between Maximum and Minimum Temperatures and Wheezing in the Midlands.

after the sixty-three day lag. After this lag the sexes' correlograms cross over and the female graph becomes progressively more positive and the male graph negative.

For minimum temperature, the correlograms share a more restricted range but the sexes seem to demonstrate opposite tendencies. The correlations at zero lag are very close to those with maximum temperatures at the same lag; -0.02 and -0.15 for males and females respectively. The male correlogram crosses zero at one day's lag and attains its positive peak at three days lag, $r = 0.07$. Its largest coefficient is found at forty-three days lag, $r = -0.16$. The male graph remains mostly negative but gains small positive values between 1-3, 30-31, 52-61, 86-87 and 98-99 days lag. The correlogram for females starts at $r = -0.16$ for zero lag and peaks at -0.19 at two days lag. It zeroes at thirty-four and sixty-three days lag but does not become consistently positive until after ninety-three days lag.

Burnie

Unlike the Hobart correlograms for maximum and minimum temperatures, those for Burnie do not demonstrate the same rising trend in cross-correlation with increasing lag; neither do they exhibit the similarities in sign and degree between the sexes shown in Hobart. The correlograms are dominated by positive coefficients with female morbidity and negative ones with male morbidity. This is in striking contrast to the associations shown in the other areas. In Hobart and in the

Midlands the correlations are of the same sign and differ only in degree (figure 4.5.5).

For maximum temperatures the female zero-lag correlation is 0.14 and the male correlation is -0.13. After four days lag the female coefficient is zero and the male coefficient increases to -0.15 with a further day's lag. After nine days lag the decreasing male coefficient reaches zero while the female coefficient increases to 0.12. The male coefficient thereafter regularly peaks at seven-day intervals at 5, 12, 19, 26, 33, 40, 47 and 54 days. The regularity of the relationship is then disturbed but seven day periods are still common between peaks. (Day-of-the-week is obviously influencing this pattern.) The largest correlation between male morbidity and maximum temperature is with a 72-74 day lag, $r = -0.34$. The female coefficient peaks with a thirty-five day lag at $r = 0.18$. After thirty-six days lag there is a marked drop in correlation that reaches -0.03 at forty-two days lag and then recovers to 0.13 by the forty-sixth lag.

The relationship with minimum temperatures are stronger than those shown with the maximum. The zero-lag correlations are 0.21 and -0.12, for males and females respectively, compared to 0.14 and -0.03 for maximum temperatures. The male correlograms with minimum temperature never reaches zero in 150 lags. Its maximum is at sixty-six days lag, $r = -0.3$. Also the seven-day period between peaks is not evident. At eight days lag the female coefficient drops from 0.21 to just above zero and then rises again to meet its maximum at twenty-seven days

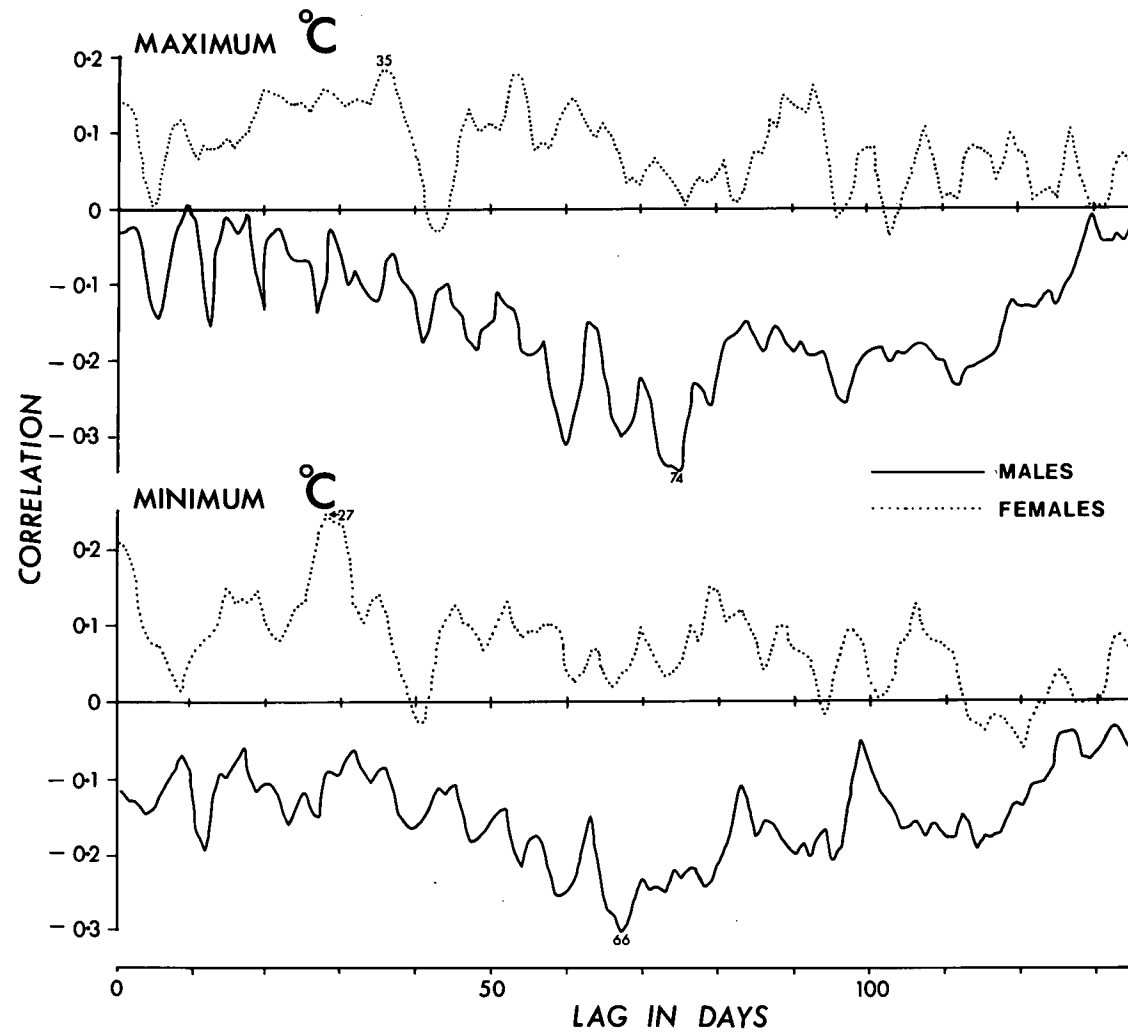


Figure 4.5.5 Cross-Correlograms Between Maximum and Minimum Temperatures and Wheezing in Burnis.

lag, $r = 0.24$. Thereafter, it drops to -0.03 on the thirty-ninth lag and five lags later regains a level of 0.12 . Further drops are demonstrated at the ninety-third and one hundred and twelfth lags.

Devonport

Devonport's cross-correlograms are the closest for the two sexes seen in any of the four areas (figure 4.5.6). In many ways they parallel the Midlands' graphs but they are more intermixed. Unlike the Midlands, but similar to Burnie, the females' correlograms tend to be more positive than the males' correlograms. At zero lag the male cross-correlation with maximum temperature is -0.18 and the female equivalent is -0.03 . This is the largest coefficient to be achieved by the male morbidity. Its positive correlation is met at sixty-four days lag, $r = 0.14$. The largest female correlation is observed with a lag of forty-six days, $r = 0.16$. The cross-correlations with maximum temperature appear to be somewhat closer than those with minimum temperature. At zero lag the male coefficient is -0.01 and the female coefficient is 0.03 . The peak cross-correlations are at eighty-one days lag for males, $r = 0.19$, and seventy-two days lag for males, $r = 0.19$, and seventy-two days lag for females, $r = 0.17$. In the females' correlograms the coefficients reached zero after only two days of lag. Male cross-correlations took five days lag and ten days lag to reach zero correlation with maximum and minimum temperatures respectively.

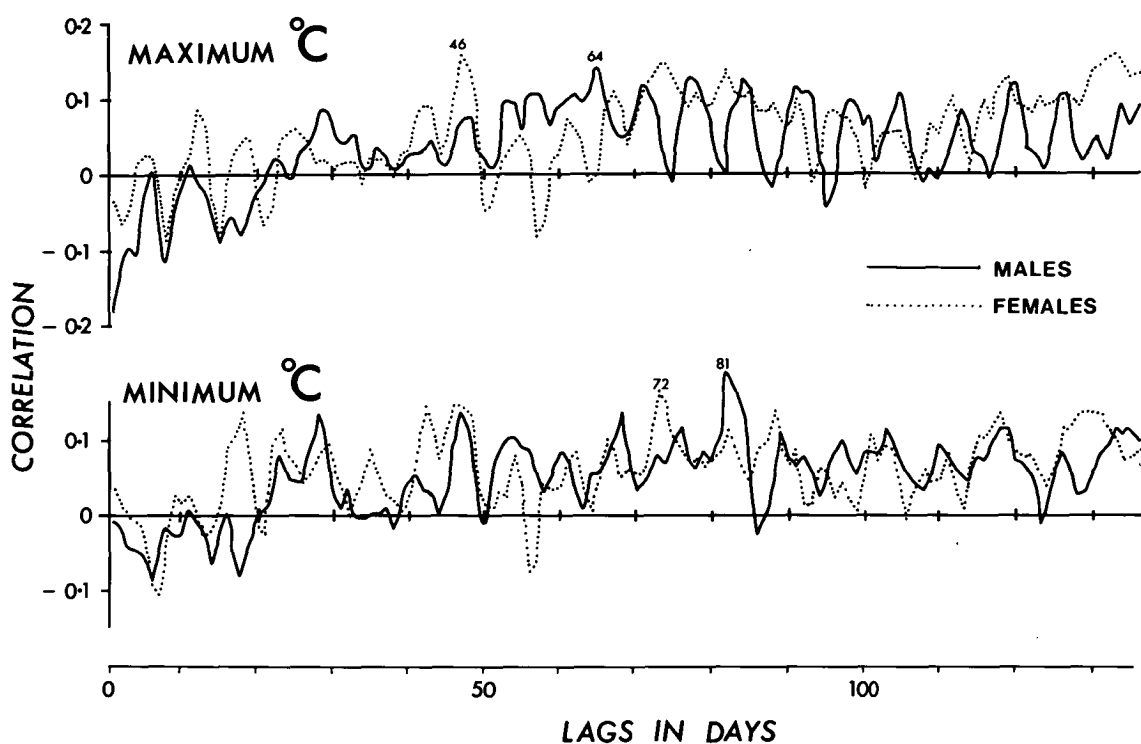


Figure 4.5.6 Cross-Correlograms Between Maximum and Minimum Temperatures and Wheezing in Devonport.

Discussion

Only a few correlograms relevant to morbidity relationships with the thermal environment were portrayed in this section. Other lagged correlations were calculated and examined for peaks and were found to demonstrate similar patterns to those already observed. Altogether 124 permutations of weather and wheezing were obtained. The correlograms for minimum temperature were similar to those for 9 a.m. wet-bulb and dry-bulb temperatures and mirror-opposite to the 9 a.m. cooling power. Correlograms for the maximum temperature were similar to the 3 p.m. wet-bulb and dry-bulb temperatures and heat-stress and mirror-opposite to the 3 p.m. cooling power. This implied that the extremes of temperature were closely correlated with the other measurements of morning and afternoon thermal environments and could, to an extent, be taken as surrogates of these other variables.

Each area gave a unique pattern. In Hobart both sexes' morbidity was negatively correlated with temperature and positively associated with cooling power. The graphs, furthermore, suggested that these relationships were related to part of a long-term process only a portion of which was represented in the correlograms. The same sort of long periods or trends were also evident in the Midlands data but were not as pronounced. The Burnie and Devonport data, within the number of days utilised, demonstrated no obvious trends but some shorter cycles. The presence of a weekly cycle was suggested by a succession of minor peaks at that period in the male correlogram with maximum temperature. This seven-day cycle was probably anthropogenic

in nature. It could have been due to either weekly changes in industrial activity or due to weekly changes in social activity. Examples could have been increased pollution in the former and exercise-induced asthma in the latter.

The correlograms support the existence of meteorotropic relationships with asthma morbidity other than those with synchronous values. In some cases, maximal correlations were with weather values that occurred some days or even months before the morbidity sequence. Male and female correlograms were not identical. They differed in degree in Hobart, were opposite in sign in Burnie and followed two opposite trends in the Midlands. The meteorotropic response was seen to be specific for each area and within each area for each sex.

Spectral Estimates

As noted earlier, individual spectra are estimated by taking the Fourier transforms of the individual auto-variances. Spectral estimates are illustrated for the four areas and their sexes separately in figure 4.5.7. The horizontal axes are scaled in days/cycle, that is, the number of days between consecutive peaks in a given wavelength. The eight spectra of the eight morbidity series are all different. Their only common factor is the dominance of a "red noise" that is, long wavelengths or trend in their periodicities.

The Hobart spectra for the two sexes exhibit the differences first detected in the auto-correlograms. Here the high persistence seen in the female correlogram is reflected in the

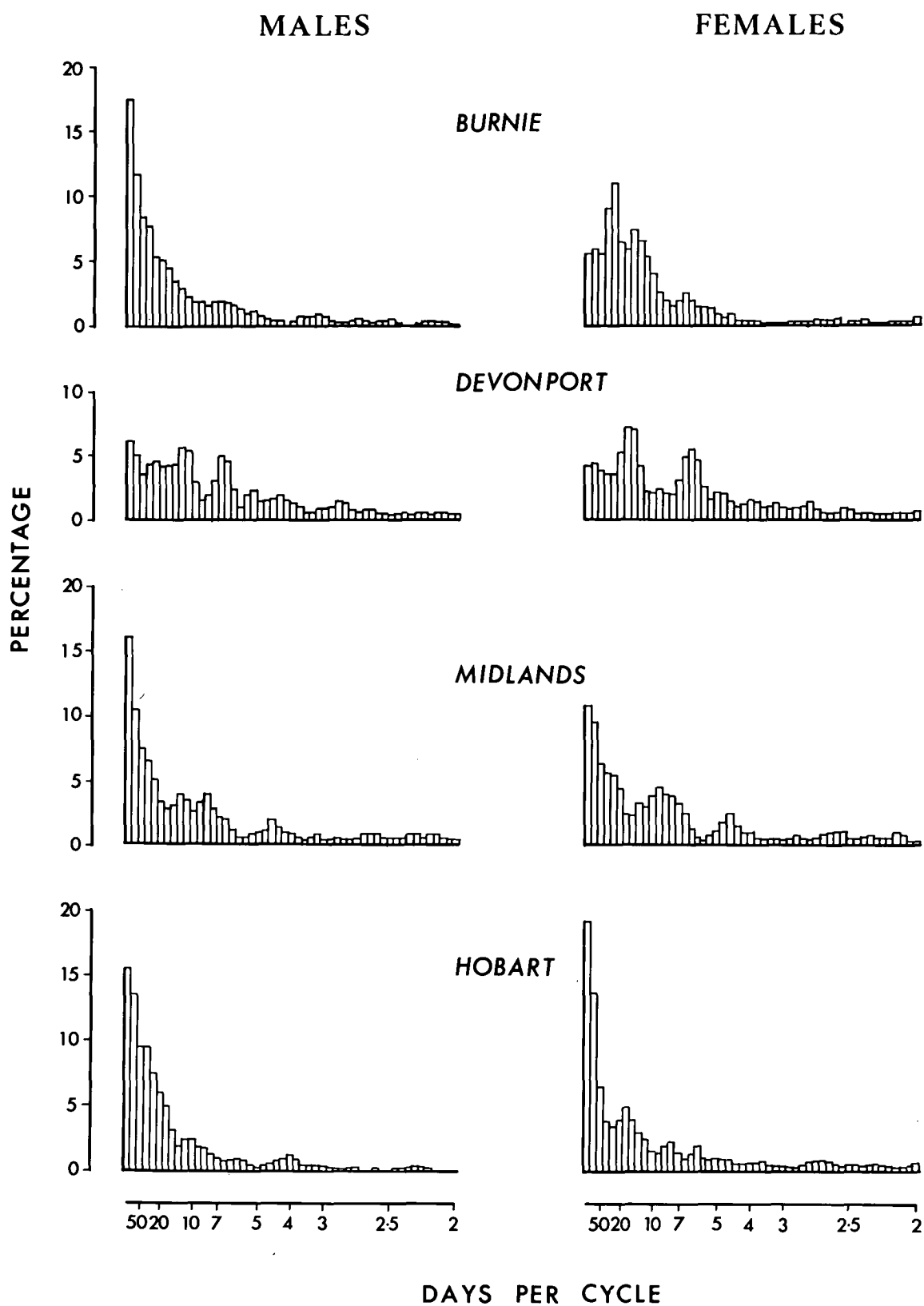


Figure 4.5.7 Wheezy Morbidity Variance Density Spectra by Sex and Area.

strong low frequency component of the spectrum. Of all the spectra, Hobart females' spectrum has the greatest proportion of its variance in cycles of 100 days or longer (36 per cent). Other points of interest from the spectrum of Hobart females' morbidity are the peak around the cycle of 16.67 days and the minor peaks for 7.7 and 5.9 day cycles. Male morbidity in Hobart is also predominantly red. Here, however, the concentration of variance remains high in a wider range of low frequencies. Fifty-five per cent of the variance, for example, is shared by cycles of twenty-five days length or longer. Minor cycles may be present with periods of ten to eleven and four days.

In the Midlands, the spectra differ from Hobart's examples. The male spectrum in this area is dominated to a larger extent than the female spectrum by low frequencies; only twenty per cent of the female variance is accounted for by cycles of a hundred days and longer compared to twenty-six per cent of the male variance. In Hobart this was the reverse. The Midlands females' morbidity spectrum possesses another twenty per cent of its variance in cycles of twenty to fifty days in length. Peaks are observed in the female spectrum at 12.5, 9.1 and 4.5 days. These obviously portray some form of harmonic regularity based on a nine-day cycle in the data. The male spectrum, in addition, has minor peaks at 12.5 and 8.3 days per cycle and an identical 4.5 days per cycle peak to the one in the female spectrum. Generally, the sexes' frequency spectra are seen to be very close. With the exception of the 16.6 day cycle in the Hobart female spectrum, the Midlands' spectra contain far more short-term variation than Hobart.

The two Northwest Coast populations show enormous differences in frequency distribution. Again, Burnie males' spectrum is very similar to that of Hobart males with about thirty per cent of its variance possessed by cycles of one hundred days or longer. Its form is fairly exponential with no peaks of any significance in the shorter frequencies. Females exhibit a very different spectrum to any observed thus far. Here, the extremely low frequencies, those cycles greater than fifty days length, are much reduced. Instead, a prominent peak is given at twenty-five days per cycle - thirty-three days per cycle and another at 14.2 days per cycle. A small waveform is also detected at 6.7-7.1 days per cycle. They are all harmonics of a seven day cycle and represent a weekly variation in morbidity.

Devonport's spectra contrast with all the others. They present the most even distribution of variance across the frequency bands. But like the Burnie females' spectrum, the red noise has been truncated, none of the cycles greater than twenty-five days in length contribute to more than about five per cent of the total variance. Each sex demonstrates two major peaks and these could represent the same seven-day cycle. In males the cycles are at 11-12.5 days and 6.7-7.1 days. In females the cycles are at 14.2-16.6 days and at 5.9-6.7 days. Additional cyclical components are present in the male spectrum at 5.1, 4.3 and 3.1 days and in the female spectrum at five days but these are of very minor importance.

Generally, no common pattern is observed between the spectra of the sexes' morbidity. Boys' spectra, though different

to, are not distinguishable from those for girls. Within each location, however, some commonality can be ascribed to both sexes. The spectra seem to grade in their extent of red noise from Hobart to the Midlands to the Northwest Coast. Hobart's spectra tend to be the most dominated by long wavelengths. This dominance decreases in the Midlands' spectra and decreases even further in the Northwestern examples. No individual frequencies stand out, either, as commonly important to all the spectra. However, both Burnie and Devonport spectra all share cycles of between 6.7 and 7.1 days in length and the female spectra for these two cities share a cycle of 14.2 days. In the Midlands both sexes possess a small cycle of 4.5 days. Both Hobart and Devonport girls display a 16.7 day cycle and both Midlands and Devonport boys display a 12.5 day cycle. Other noteworthy cycles occur at ten to eleven days in Hobart males, 9.1 days in Midlands females, and twenty-five to thirty-two days in Burnie girls. Many of these estimates could be interpreted as seven-day cycle harmonics.

Throughout this work analyses show differences in morbidity patterns between the sexes and between residential areas. The spectral analyses demonstrate the relative importance of the several constituent frequencies in the morbidity series for eight groups of asthmatics. Attention is drawn to the dominance of long-term cycles and trends and the minor amounts of variance contained in short-term fluctuations. Because of the low proportions of variance accounted for by the short wavelengths in the morbidity spectra any explanatory power will be restricted

to medium and longer length frequencies. To examine this proposition several cross-spectral analyses are performed for the several pairs of weather and morbidity series.

Cross-Spectrum Estimates

Just as it is possible to estimate the spectrum by a Fourier transformation of the auto-covariance function it is possible to obtain the cross-spectrum from a Fourier transformation of the cross-covariance function. As output from the cross-spectrum analysis one obtains the two spectra, the amplitude of the cross-spectrum, the amplitude of the real (in phase) and imaginary (out-of-phase) parts of the cross-spectrum, the phase spectrum and the coherence spectrum. The morbidity spectrum and its coherence and phase relationship with the weather spectrum are the only necessary pieces of information to describe the normal bivariate frequency distribution.

The total number of cross-spectra examined was unmanageably large and only one set of morbidity weather cross-spectra are presented here for illustrative purposes. Figure 4.5.8 presents the coherence estimates for the cross-spectra between each area and sex specific morbidity series and minimum temperature. Coherence is equivalent to the square of the correlation coefficient for each frequency band in the two spectra and hence measures the strength of relationship between each frequency in the two series. Because it is a squared number, however, the sign of the relationship is unknown. The direction of the relationship has to be obtained from the phase spectrum.

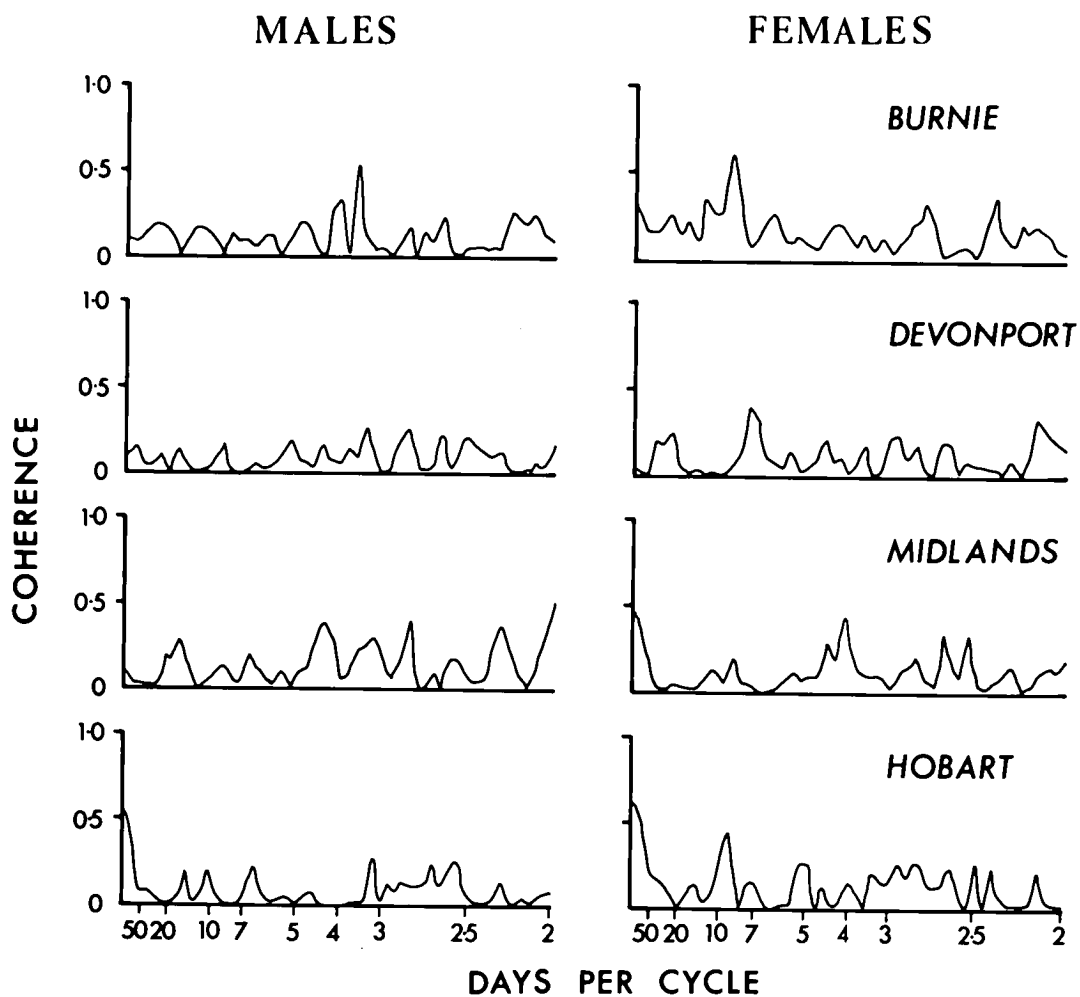


Figure 4.5.8 The Coherence Spectra for Wheezing and Minimum Temperature by Sex and Area.

Two waveforms that are in phase are perfectly positively correlated. Two waveforms half out of phase are perfectly negatively correlated.

The plots of coherence in figure 4.5.8 need to be examined with the morbidity spectra in figures 4.5.7. It is observed that both Hobart sexes have high coherence in their long wavelengths. This is also true of Midlands girls. Resolution was poor at these frequencies because of the short length of the base series (one year) and those estimates, however interesting, have to be ignored. Many of the coherence plots contain peaks in the region of 0.4-0.6, forty and sixty per cent explanation respectively. Such peaks indicate the presence of cyclical components that are shared by the weather and morbidity series.

For example, in the coherences of Burnie girls and Hobart girls with minimum temperature peaks are seen at a nine-day cycle. This suggests that there is a relationship between female morbidity in these two locations and minimum temperatures and that this relationship follows a nine-day cycle. An examination of phase relationships in the two areas gives the phase in the Hobart relationship as 0.55 and in the Burnie relationship as 0.96. These represent almost exact negative and positive correlations respectively. This demonstrates that there is a nine-day periodic relationship held in common between female asthmatic morbidity and minimum temperature in each of the two areas but that in each area the relationships/reactions are opposite.

How important is this effect? To answer this question one has to inspect the morbidity spectra to see how much of the morbidity variance is accounted for by this frequency. It is seen that this amounts to 2.5 per cent of the Burnie and 1.5 per cent of the Hobart females' morbidity variance. As the coherence is only about fifty per cent the actual explained variance in the morbidity spectra is 1.3 and 0.8 per cent respectively. The many other cross-spectra when similarly perused do not give any explanation greater than one or two per cent for an individual frequency estimate. So, although short-term cyclical components are found in the relationships between wheezing and weather factors, they are generally of low explanatory value. The existence of season fluctuations in asthma morbidity has long been recognised. Cycles that account for larger amounts of variance, and hence explanation, are probably of this scale of wavelength. The importance of these quasi-seasonal, annual and trend-like cycles can only be assessed by the analysis of many years of daily data. The only short-term cycle in morbidity that is detectable is a weekly cycle in the Northwest populations. This is not a meteorotropic effect. Short-term effects may not be cyclical at all but may resemble random shocks.

4.6 Summary

The data base used for analysis in this section was specifically collected for the purpose of studying meteorotropic reactions in the incidence of wheezing in different geographic populations. Unlike the majority of previous studies of "asthma" and the weather that used casualty or hospital admissions as a morbidity index, this study emulated Tromp's approach instead. His research was based upon "captive" populations of asthmatic schoolchildren living in a residential school in the Netherlands. This research, instead, monitored the incidence and severity of wheezing in populations of schoolchildren from the 1971-72 birth cohort of Tasmanians who lived in four different and spatially defined parts of the state. In this way, age, sex and residence were controlled for and the full range of wheezy-breathing was measured. In other previous studies neither age nor sex nor residence nor severity have been able to be taken into account.

Analysis demonstrated differences both between the sexes and between the areas in their incidence of wheezing during the course of a year. This provided yet more evidence to that already accumulated upon these meteorotropic differences from earlier sections of this work. Once these patterns in the morbidity series had been established the thrust of analysis was to explore the associations between the wheeziness and various factors of the atmospheric environment. Various correlation analyses were performed at several time scales and for several types of seasonal and synoptic situations found in Tasmanian weather.

The correlations were found to vary both with the length of time period over which they were calculated and with the type of weather systems. When moving-correlations were calculated the coefficients were observed to vary continuously over time, sometimes in a quasi-cyclical pattern. Extreme variations in correlation were related to strong weather events. In most cases the correlation constellation was interpreted as describing a reaction to the totality of thermal stress and the variation in relationship was hypothesised as being related to long-term physiological adaptations or fluctuations concerned with the process of acclimatisation.

The nature of the changing relationships and the presence of what appeared to be cyclical components in the data gave rise to the idea that the morbidity-weather relationship was periodic. The changing values of correlation coefficients over time was to be explained by different frequency components, possessing different correlations, interfering with each other and producing a complex moving-correlogram.

The initial analysis in the frequency domain was the construction of the variance-density spectrum for each of the morbidity series. When these were examined, again, all were different although some areas gave fairly similar spectra for the two sexes' morbidity series. The majority of the spectra contained red noise and were dominated by long-wavelengths and trends. Many, however, contained significant peaks at certain shorter frequencies, and these peaks represented waveforms existing in the morbidity series.

To see if these cyclical components in the majority series were related to similar components in the weather series, numerous cross-spectrum analyses were carried out. Although the coherence estimates for particular frequency estimates were occasionally very high these did not involve the dominant middle-to-short frequencies which arose in the morbidity spectra. Short-term periodic components common to both the weather and morbidity series never accounted for more than two per cent of the morbidity variance. Explanatory variance tended to be concentrated in the longer wavelengths but these were poorly resolved due to the short length of the morbidity record and confidence could not be placed in their coherence estimates.

The analyses in the frequency domain indicated the presence of short-term cyclical components in the morbidity series but also demonstrated their negligible associations with weather factors. Any weather wheezing relationships were probably to be found in the longer, quasi-seasonal cycles present in both types of data. Of all the analyses the correlation exercises were most rewarding. These, and in addition the lagged cross-correlations, performed as preliminary stages in the cross-spectrum analyses provided the basic building material for the construction of models of asthma morbidity based on weather elements. In the next section such models will be built and tested for efficacy on the remaining seven months of data that were collected specifically for such a purpose.

Section 4

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SECTION 5

CONCLUSIONS

5.1 Introduction

The general aim of this work was to examine the spatial and temporal variability of asthma morbidity in Tasmania. To this end three populations of asthmatics have been investigated and their morbidity status has been assessed for spatial patterns and changes over time. The results of these studies have enabled the three null hypotheses stated in Section 1.3 to be rejected.

These were that:

1. no significant variation existed in the spatial distribution of wheezers or of clinical subsets of asthmatics/wheezers in the State of Tasmania,
2. no significant variation existed in the temporal distribution of wheezing episodes in the total population of asthmatics or in subsets of asthmatics defined by clinical symptoms, sex, or residence,
- and 3. that no significant relationship existed between any wheezy morbidity series and any physical environment series measured over the same time period.

The probability maps in section two demonstrated that significant spatial variation did exist in the distribution of the total and of various sub-sets of wheezers. Results from the analyses of both the hospital admissions in section three and the wheezy episodes in section four clearly illustrated the extent of

temporal variability. Significant variations were observed at many time scales from annual to those of a few days. Furthermore, temporal variations were shown also to exhibit spatial variations. Significant temporal variation was not limited only to the domain of time but also to the domain of frequency. Morbidity series from different areas possessed different cyclical components.

Significant correlations between morbidity series and physical environment series were obtained partially from section three analyses of hospital admissions but mostly from those in section four on episodicity. Correlations between weather and morbidity were often very high. Both the magnitude and significance of the correlation coefficients were to a large extent dependent upon the environmental homogeneity of the days upon which the calculations were based. Annual correlations based on 365 days differed from those for seasons based on about ninety days and these differed from those based on synoptic classifications which may have occurred on as few as twenty days.

In addition to the rejection of these null hypotheses, the general aims and objectives of the study included the generation of models of asthmatics' environmental risk. At one scale this has already been achieved. The several maps in section two for example, indicated areas where surpluses and deficits of wheezers with differing grades of symptoms or severity resided. Children's health status has been regarded as a sensitive indicator of environmental quality. Prevalence maps of a severe wheezing history or spirometry variable thus provided a static model of long-term exposure to various

environments. Spatial clustering in the prevalence of persons with poor spirometry performance, for example, delineated areas of "high risk" and *vice versa*.

Some of these cartographic models are reviewed later in section 5.2. They attempt to answer the question of "If you are a seven-year-old liable to wheezing where in Tasmania is the best place to live for respiratory health?" A caveat must be made before their interpretation as to the applicability of their patterns to the wider population. They are based upon the respiratory health status of seven-year-old children. It might be argued that the characteristics of this population cannot be justifiably extrapolated to other ages, but, bearing in mind the sensitivity of this age group to atmospheric quality and their lack of occupational exposure, their use as an indicator of ambient exposure to environmental risk can be supported.

These cartographic models are similar to maps of climatic regions; they indicate areas which in the long-term, or on average, influence towards certain degrees of morbidity in much the same way as deserts or polar regions reflect the long-term or average degree of precipitation or radiation. Static prevalence models, however, only represent half of the environment-morbidity complex; just as climate maps only indicate long-term, overall averages and ignore weather events. There is an obvious need to supplement the maps of long-term environmental exposure with dynamic models of short-term meteorological hazards. Again, using the analogy with climate the important

events in a desert environment are different in nature to the important events in a polar environment. Because of the difference in stress events, each type of morbidity region requires a separate model of meteorological risk. Such models are generated using regression methods in section 5.3. These are then tested for efficacy upon the seven months of data collected from June 1978 to December 1978. Together, the maps and regression models fulfil the original objectives of this study. The final sub-section, 5.4 acknowledges its limitations and indicates the areas for further study.

5.2 Static Models of Environmental Risk to Asthmatics- Wheezy Breathers in Tasmania

Introduction

Because of the cross-sectional nature of the 1968 asthma survey, complete geographic coverage was achieved. This enabled the production of morbidity prevalence maps of unusual accuracy. Any spatial clustering observed in the cartographic analyses should be free of the recording and diagnostic artefacts common to some surveys and the probability distributions over space are, therefore, believed to represent a good estimate of the true population variability about the mean or state norm.

Common to both the asthma survey analyses and the hospital admissions analyses were differences due to the sex of the subjects. Female wheezers of varying types and degree, consistently demonstrated different, sometimes opposite, spatial concentrations to their male counterparts. The seasonal patterns obtained from hospital admissions were also different. Male seasonality was dominated by an Autumn peak, female seasonality by a Spring peak. This contrast was reinforced by the correlation analyses from section four. The two sexes demonstrated different correlation constellations for the seasons and also for shorter periods. Such differences have not been discussed in the literature, on the contrary, a lack of difference has usually been noted. The influence of sex, therefore, could not be ignored. On this basis, separate maps have been produced for males and females.

Wheeziness

The history of wheezy-breathing was one of the two major symptoms that the 1968 survey used as the basis for classification. Altogether, 19.1 per cent of the boys and 13.2 per cent of the girls had experienced this symptom. Their spatial distributions, first illustrated in figure 2.2.1, are given again here in figure 5.2.1. No statistically significant excesses of wheezers were detected but for each sex an area was found that contained only half the expected population. For males this was the Huonville-Channel area in Southern Tasmania. Here, the prevalence was only ten per cent of the population compared to the average of nineteen. Another "healthy" area for males was the Northeast quadrant of the state. This area with a prevalence of twelve per cent was significantly lower than the norm at $p \leq 0.05$. An area with half the expected female wheezers was located between Devonport and Burnie on the Northwest Coast. Here a prevalence of 6.3 per cent could be compared to the 13.2 per cent average. Another area in the central Midlands gave an even lower proportion, 4.9 per cent, but because of low numbers this was only significantly lower than the norm at $p \leq 0.05$. If the two areas significant at $p \leq 0.01$, were considered on their own it was noteworthy that a natural female sanitorium existed on the North Coast and a male equivalent existed on the South Coast.

Wheeziness alone describes little else than the potential for reversible airways obstruction. Many children have a symptomatic wheeze only once in their lifetime associated with an

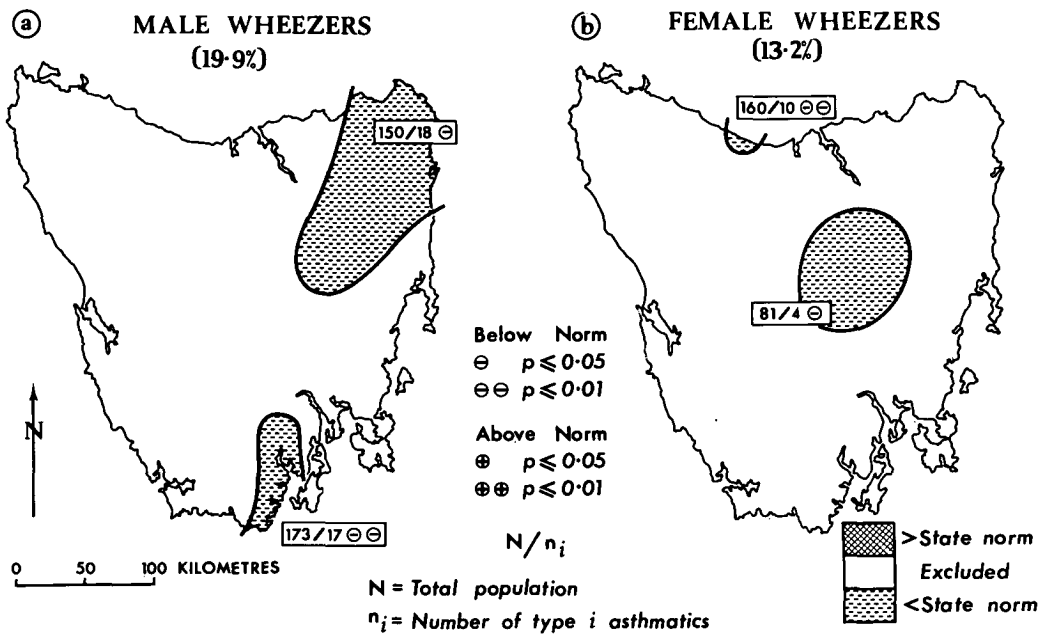


Figure 5.2.1 The Spatial Distribution of Wheezers by Sex.

acute respiratory infection. These cases obviously would not be described by a doctor as "asthmatic". Given a sufficient insult, everyone is a potential asthmatic on this basis. Section two contains maps of many different symptoms and classifications. This plethora of maps may upon first inspection present anything but a clear impression of the geographic variation of wheezy breathing. In part, this is due to the chronic and heterogeneous nature of the complaint; there is no simple categorisation of when wheezy breathing becomes asthma. Severity, gradation and prognosis are all dependent upon several factors. In fact, mild forms of symptomatic wheeze probably have negligible effects upon long-term health status. Thus, the distribution of mild wheezers can be seen to confuse the delineation of the healthful from the hazardous areas on the maps because they are based on the sole criterion of wheezing. In order to detect unhealthy environments one needs a quantitative rather than a qualitative measurement of respiratory efficiency. Fortunately, this information is available in the form of spirometry readings. Whatever the cause or pathway that provoked an individual to become wheezy, these accurately record the effects.

Spirometry

The spirometry variables have been standardised for height and mapped in section 2.5, figures 2.5.2. - 2.5.5. Spatial distributions of the four inter-correlated measurements were very similar. This was expected. In fact, such minor differences

as did exist were thought to be due to the slightly different numbers falling above and below a half-standard-deviation from the norm. If quartiles had been used, for example, employing identical numbers and populations, the same spatial patterns would most probably have arisen. This absolute difference in numbers was the most probable explanation for the minor differences in distributions between the figures of individual spirometry variables and the composite spirometry dimension constructed by factor analysis in section 2.7. Figure 2.7.2 illustrated the factor scores above and below one standard deviation from the mean. The numbers categorised into these classes were naturally much smaller than in the single variable maps. The factor maps, therefore, portrayed populations of greater extremes of morbidity than the spirometry maps.

From the four spirometry maps, one can attempt to form a composite map of good scores and poor scores. Figure 5.2.2 achieves this objective by overlapping the areas from the four individual maps that possessed an excess of a particular score. The darkest areas represent four overlays and the white areas represent average scores between ± 0.5 standard deviations from the mean. Four maps are given in the figure to classify both male and female and both good and bad areas. Bad spirometry environments differ for the two sexes. For males the worst areas are the centre of the North Coast and the city of Hobart, and the Lower Derwent Valley. From the worst area on the central North coast a band of low average values extends to the

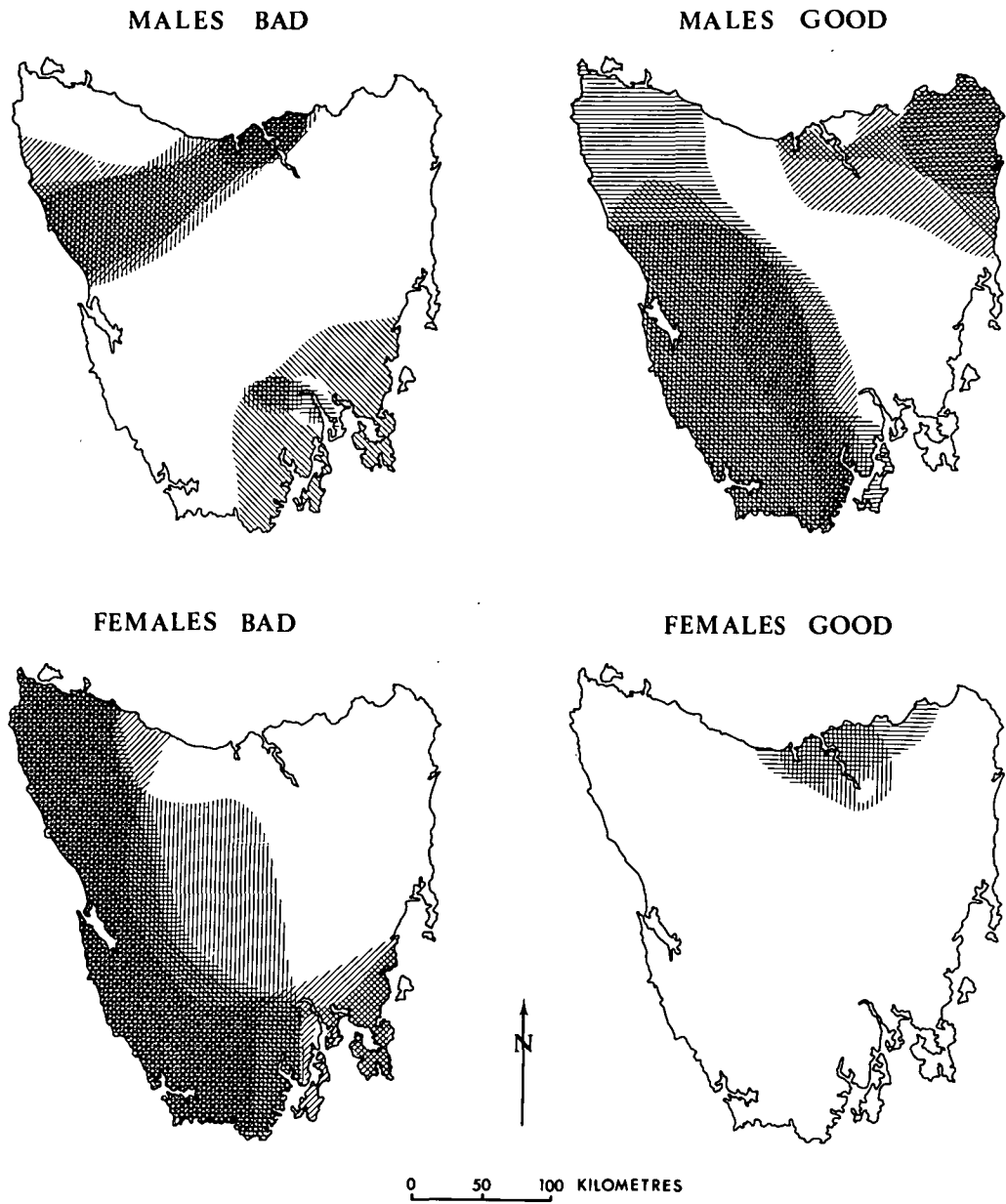


Figure 5.2.2 Composites of the Spirometry Maps.

West Coast. In females the bad areas start in the extreme Northwest and extends South and then East avoiding the Hobart metropolitan area. There is a slight overlap between the sexes' good environments. This occurs in the North central part of the State. Healthy male values are concentrated in the extreme Northeast but grade westwards to Devonport avoiding Launceston and the industrial towns at the mouth of the Tamar. The healthy female area occupies most of the Tamar estuary but does not extend as far to the East as the male pattern. In contrast to the unhealthy area for females, the males possess another healthy area that is focussed upon the Huon and interior regions of the state discussed earlier in section 2.9. This declines in a gradient slowly to the West and more sharply to the North.

It is interesting to compare these composite maps with the spirometry factor maps in figure 2.7.2. Figure 5.2.3 illustrates figure 2.7.2 with only the positive excesses shown. That is, the good and bad areas are indicated only by the surpluses of good or bad scores on the spirometry factors not by the deficits. In this way a region lacking in good scores is not interpreted as being bad and a region lacking in bad scores is not necessarily interpreted as being good. Because the factor maps are based on smaller, more-extreme groups they illustrate greater degrees of health and hazard to asthmatics. Areas in 5.2.3 which could be interpreted as hazardous to male asthmatics correspond closely to those in figure 5.2.2. They include an area surrounding Devonport in the middle of the

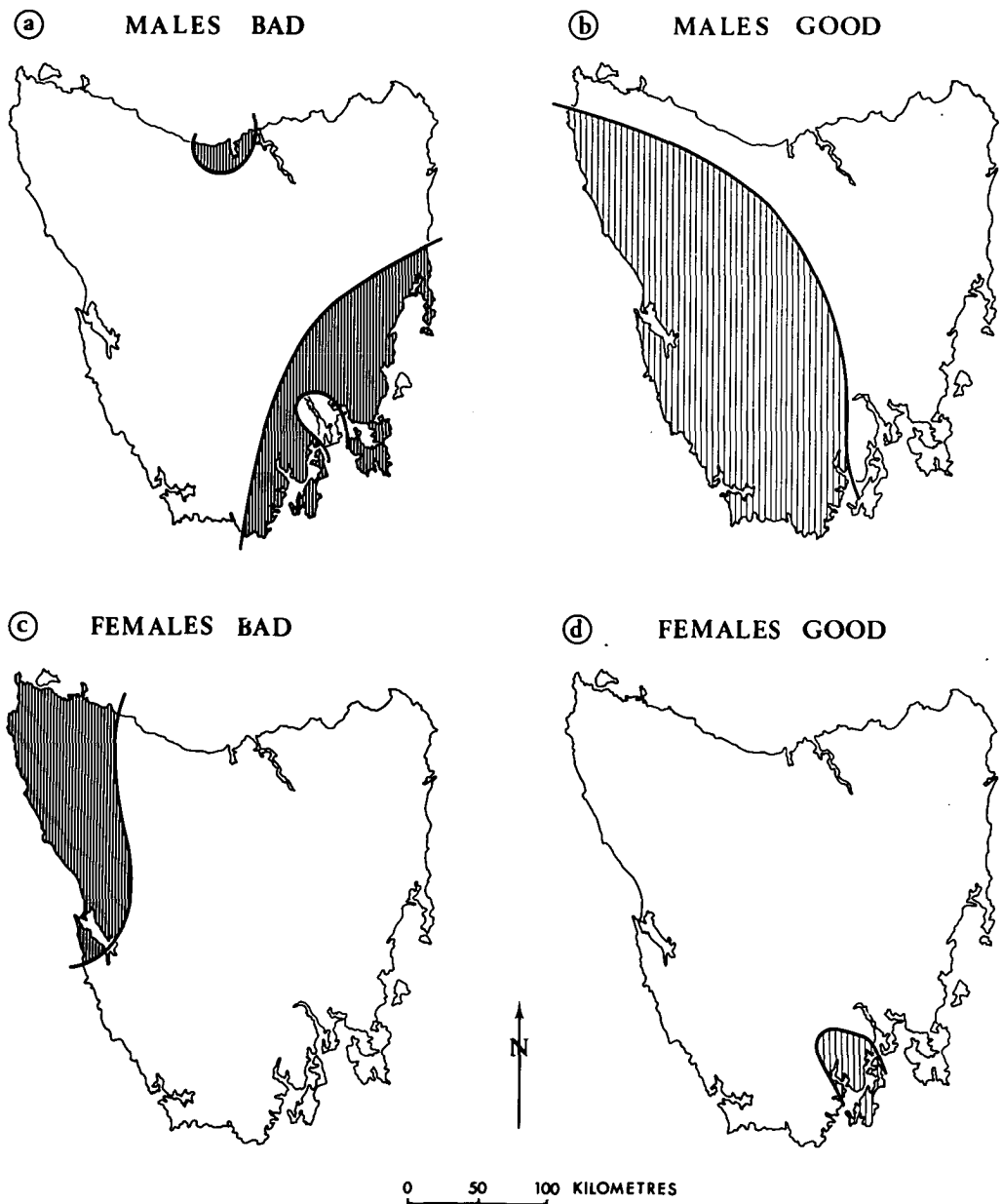


Figure 5.2.3 Good and Bad Areas for Asthmatics Based Upon the Areal Concentrations of Extreme Scores on the Spirometry Factor.

North coast and an area surrounding Hobart that embraces the entire South-Southeast coastal quadrant. The high risk area for females is much contracted compared to the pattern in figure 5.2.2, it includes the extreme Northwest and West Coast. The healthy environment for male asthmatics shown in figure 5.2.3 parallels that found in figure 5.2.2. It includes most of the Western half of the island but not the Northwest Coast. This is the area of highest rainfall in Tasmania. Female asthmatics with 'good' spirometry readings differ in their pattern in this figure from their pattern in figure 5.2.2. Instead of being concentrated on the North Coast, they are clustered in the Huon-Channel area in the far South. This cluster is closely similar in distribution to the deficit of male wheezers shown in figure 5.2.1, a healthy area by virtue of its absence of complaint. Taken together, the spirometry 'factor' maps must be considered as superior to the 'composite' maps. They separate 'good' from 'bad' areas more clearly.

Summary

The three figures illustrated here are but a small selection of those available in section 2. Different types and degrees of wheezy breathing and asthma symptoms demonstrate a variety of individual spatial patterns. These, however, usually represent only one facet of the complex syndrome and do not make any general information available that is not restricted to seven-year-old children. The subject matter of the figures includes the phenomenon of wheezing and the

variability of lung function. These two indices are general indicators of respiratory health. The first indicates the presence of reversible airways obstruction, and the second measures the effect of this condition and the influence of environmental exposures and insults. Because of their "meaningfulness" and the ease of interpretation these variables constitute the most useful parameters of respiratory health that are available. Their variations can be extrapolated to the wider community. This is one of the benefits of child health studies; for children can be used in much the same way as canaries were used in mines, as environmental sensors.

5.3 Dynamic Models of Environmental Risk to Asthmatic - Wheezy Breathers in Tasmania

Introduction

This summary exercise represents a natural extension of the analyses from section four. It uses the same data base, the specially collected wheezing incidence from geographic samples of the 1971-72 birth cohort, and uses the findings from the correlation analyses in section four as input to the modelling process. In all, nineteen months of data upon the incidence of wheezing were collected in four locations in the state. The first twelve months' of data were analysed in section four. This year of daily data is used again here for multiple regression analysis of the effect of weather variables on wheezing for the separate sexes in each area. The remaining seven months' of data are available for testing the efficiency of the regression equations for future use.

The objectives that underlaid the analyses in section four were to elucidate the interrelated mechanisms acting between wheezing and weather and to discover the time scales at which these were operating. For these purposes, the time series were analysed both in real time and in their frequency components. Both of these perspectives gave an initial impression of wide variation between the two sexes and between the four areas. Such variation precluded the construction of a general model for all individuals regardless of sex or residence. It became apparent that empirical models would have to be fitted to each sex-residence and weather category.

Originally, it had been intended to produce an autoregressive-moving-average model based upon spectral and cross-spectral estimates. Unfortunately the lack of significant cyclical components in the shorter frequencies and the concentration of variance in seasonal and longer wavelengths prevented any useful modelling based on the lengths of the small series that were available. Furthermore, it was suspected that some of the morbidity series were not stationary, that is, they possessed trend and their variance was not constant. The children in the study were aged between six and seven years. During the nineteen months of surveillance some individuals enjoyed a decrease in the severity, and/or frequency, of their symptoms. This remission around the age of seven was also observed in the hospital admissions statistics. Between the ages of six and ten the casualty attendance rates at public hospitals dropped significantly. The trend could be confidently interpreted as a real phenomenon and not as an artefact of the survey methodology.

The presence of cyclical components in the morbidity series was of interest, especially the existence of a seven-day cycle. This cycle and its harmonics, however, only accounted for a minor proportion of the total variance in the spectrum in some of the sex-location categories. The most noticeable of these were the Northwest Coast cities especially Burnie. Seven-day cycles had obvious anthropogenic origins. They could have been due to indirect results of the working week. Weekly

cycles could have been linked to variation in industrial or vehicular pollution. Alternatively, they may have been the result of social activities again structured by the working week. For example, there is an increased possibility of exercise-induced asthma on the weekends when playing sport, running on the beach or going for family outings. The existence of these cyclical components in the data that are probably not related to meteorological variables would have contributed noise to any meteorotropic model. This was considered negligible because of their modest contribution to the overall variance.

The frequency analyses unearthed very little in regard to the relationships between wheezing and individual weather phenomena. The preliminary steps of cross-spectral analysis, that of cross-correlations with lags, however, were of value. Correlograms between weather and morbidity for the several areas showed which lagged relationships possessed the highest correlations. Such lagged variables were therefore available as input to the models in addition to the concurrent values.

A large part of section four had been devoted to the calculation of correlations between the various weather and morbidity permutations. These had been repeated for different time scales; annual, seasonal and monthly and for several synoptic weather classifications. Correlation constellations were observed to change both with the length of the time series and with weather types. The differences between the seasons were considered to be due to differing proportions of synoptic types at different times of the year. This proposition was

supported by the examination of moving cross-correlations. These illustrated how the relationship between any pair of morbidity weather variables changed over time. Significant periods of high cross-correlation were related to the occurrence of particular types of synoptic weather situation. When calculated over a longer period of time these high correlations were usually reduced to close to zero for often in the course of 6-8 weeks the coefficients had changed from highly positive to highly negative and had cancelled each other out.

The result of this approach to the data was to highlight both the importance of particular weather systems and the importance of population sensitivity to the understanding of fluctuations in wheezy morbidity. The several sub-populations all gave different moving-cross-correlations but an examination of the correlation constellations at the different peak correlation periods showed the same basic relationship to temperature and atmospheric cooling. Each group was seen to possess reactions to meteorological insults but these reactions were not synchronous. The response to stress obviously was not only dependent upon the nature and degree of the stimulus but also upon the nature and degree of sensitivity of the morbid population.

This sensitivity or "morbidity limit" was seen to be modified by past meteorological conditions and by the slowly changing seasonal regime. Children living in different areas obviously enjoyed different microclimates that affected their morbidity limit thresholds to different degrees. The several populations' reactions to a common, major weather event were

therefore not identical. Populations with a high average threshold did not react as strongly as those with low average thresholds. Furthermore, the individual morbidity limits could have been modified by a variety of non-meteorological factors for example, passive smoking, the ambient levels of air pollution, exercise and the use of prophylactic drugs. (INTAL).

This concept of a variable, acquired resistance to stress makes the modelling of morbidity on the basis of fluctuations in the several stressors very difficult. An empirical model usually can be fitted to virtually any relationship. Problems arise, however, when an attempt is made to predict future values, for the variability in morbidity limit is equivalent to changing the rules upon which a model is based. Bearing these reservations in mind, an attempt is made to produce models of morbidity based upon atmospheric elements for each area-sex specific group.

*Multiple Regression Models of Wheeziness Based Upon
Atmospheric Quality*

For each area and sex the weighted morbidity index was the one chosen for modelling. In these the daily incidence had been adjusted for severity; a severe attack that resulted in an emergency visit to hospital was considered to be more important than an episode of mild wheezing. The weighted index was to be the dependent variable in each analysis. The independent variables consisted of the generally-available, current, daily weather variables and the important lagged variables gleaned

from the cross-correlograms in section 4.5. Hobart, in addition, had partial coverage for daily acid-gas levels.

The computer programme chosen for analysis was the stepwise inclusion model from S.P.S.S. Version 7. In this method the best explanatory variable is chosen first and then the next and succeeding variables are included on the basis of the greatest additional contribution to explanation. In this way the "best" explanatory variables were selected. Actually, the matrix of independent variables was sometimes strongly inter-correlated. The inclusion of highly inter-correlated variables in the same regression equation, however, would have been unlikely because of the small amount of additional explanatory variance likely to be gained from such variables. In any equations that were obviously strongly affected by multicollinearity the regressions were recalculated using only the dominant variable from the particular constellation. The correlation matrix given at the start of each analysis assisted in the detection of such relationships. Each final multiple regression model was then tested for predictive skill on the seven months of data from June 1977 to December 1977.

Like the correlation analyses in section four, regression analyses were conducted for each location and sex category at several time scales. At the annual and seasonal scales the explanatory power was low, usually between ten and twenty per cent. The reason for this was discovered to be due, again, to the importance of synoptic processes and their

relative frequencies over time. To illustrate this, an example was made of the best multiple regression model, based on the whole twelve months' data, that of Hobart girls morbidity. This model explained twenty-eight per cent of the variation in the girls morbidity. The predicted morbidity HG (Hobart Girls) was calculated thus:

$$\begin{aligned} \text{HG} = & 2.3 - 0.165 \text{ MINIMUM TEMPERATURE} \\ & (26\text{BD}) + 0.162 \text{ 9 a.m. COOLING} - \\ & 0.656 \text{ MAXIMUM TEMPERATURE (11DB)} \\ & + 0.619 \text{ 3 p.m. COOLING (1DB)} (\pm 1.7). \end{aligned}$$

The number followed by DB in parentheses indicated a lagged variable, for example (26DB) means twenty-six days before. The F-ratio was 33.8 which was significant at $p < 0.001$ and r^2 was 0.28. The standard error was 1.7. The significant F-ratio meant that this regression was not due to chance and the r^2 value meant that twenty-eight per cent of the variation in morbidity was explained by the weather variables in the regression equation. The remaining seventy-two per cent was presumably due to unknown and chance factors.

In an attempt to discover why the explanation achieved was less than one third of the total variance a plot was obtained of the residuals from the regression against their sequence. The horizontal axis of figure 5.3.1 was therefore made to represent time. The first residual plotted was for the 27th June 1977. Fifty sequential days of standardised residuals were plotted in this figure from June 27th until August 16th. Upon inspection, one noticed the oscillatory

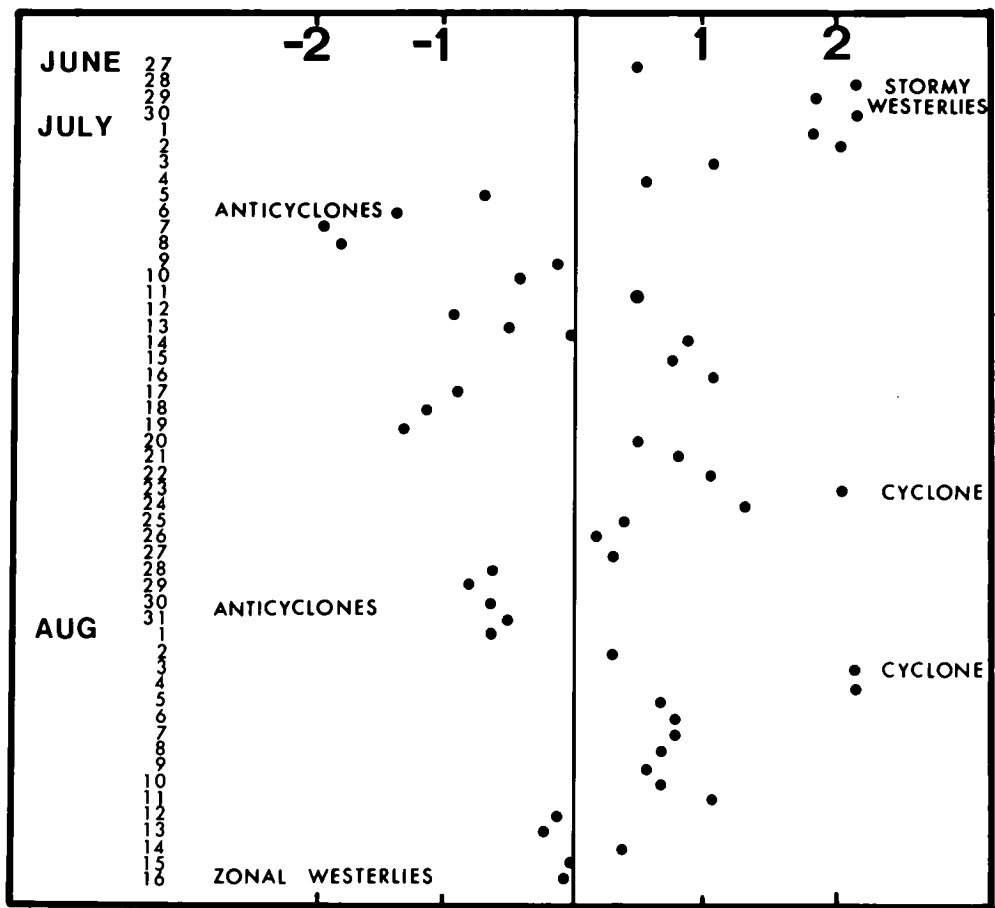


Figure 5.3.1 Residuals from the Regression Line Illustrating the Effect of Synoptic Situations.

nature of the residuals as they varied from strongly positive to strongly negative standard deviations from the regression line. This cyclical pattern could have suggested the presence of serial correlations in the data that were not due to the weather elements.

To examine this more closely the extreme residuals' dates were related to the appropriate weather charts for those occasions. An interesting pattern resulted. The highly-positive residuals occurred on days of extreme atmospheric cooling. These days were dominated by either stormy-westerlies or by strong cold fronts associated with cyclonic activity. Negative residuals, on the contrary, were associated with anticyclonic conditions, sunny calm days. Between the 6th and 10th of August the residuals were extremely small. During this time Tasmania was dominated by zonal-westerlies, one of the commonest weather patterns in Tasmania. The regression equation was obviously biased toward the average weather experience, the zonal-westerly; and the pattern of residuals was related to the cycles of strong cyclones and anticyclones that disturbed this trend. Because of this, and other examples, synoptic conditions were accepted as the basic meaningful meteorotropic unit.

Further regressions are therefore based upon the number of days of occurrence of the various synoptic classes used earlier in section four. The findings are arranged by the nine synoptic classifications and then by the four geographic areas to aid discussion. Only those regression equations which, both

in totality and in their constituent variables, are significant at $p \leq 0.05$, are presented for discussion. Both the level of explanation that is attained and the predictor variables that are included in the equations change from place to place. First, are considered anticyclonic patterns.

Anticyclone over the Great Australian Bight

The highest proportion of explanation that was achieved under this synoptic situation was Midlands girls' morbidity, fifty-six per cent. This was twice that of Hobart girls based on twelve months' data. The Northwest Coast's and the Midlands boys' morbidity indices all had less than twenty per cent explanation in their regressions. The equations for Hobart were:

$$\begin{aligned} \text{HB} &= -64 - 0.161 \text{ MINIMUM TEMPERATURE} + \\ &\quad 0.167 \text{ RAIN} + 0.066 \text{ 3 p.m. PRESSURE } (\pm 1.84) \\ \text{HG} &= -3.25 + 0.396 \text{ 3 p.m. COOLING (26DB)} + \\ &\quad 0.226 \text{ DIURNAL RANGE} - 0.879 \text{ MAXIMUM TEMPERATURE} \\ &\quad (11\text{DB}) + 0.15 \text{ 3 p.m. COOLING} - 0.343 \text{ GUST SPEED} \\ &\quad + 0.266 \text{ EVAPORATION } (\pm 1.57) \end{aligned}$$

Hobart boys (HB) gave an r^2 of twenty-six per cent the girls r^2 value was forty-eight per cent. During times when an anticyclone is stationed over the Bight Tasmania is often subjected to a single cold front associated with a southerly trough that demarcates two successive high-pressure centres. The regressions probably describe the effects of such cold fronts. The female regression, particularly, is heavily influenced by atmospheric cooling, rain and wind.

In the Midlands only one variable was included in each regression. They were as follow:

$$\text{MB} = 0.247 + 0.382 \text{ MINIMUM TEMPERATURE} \\ (3\text{DB}) (\pm 0.79)$$

$$\text{MG} = -30.68 + 0.031 \text{ 3 p.m. PRESSURE } (\pm 0.84)$$

The males possessed an r^2 value of only six per cent but the females obtained the highest r^2 for any of the regressions in this synoptic class, fifty-six per cent. In this equation the atmospheric pressure at 3 p.m. explained over half of the variation in the Midlands girls' wheezing.

On the Northwest Coast the explanatory power of the equations was better than that for Midlands males but was less than twenty per cent. In Burnie, it was impossible to calculate a significant regression for female morbidity. The male equation was as follows:

$$\text{BB} = -4.3 - 0.12 \text{ MINIMUM TEMPERATURE} + \\ 0.023 \text{ 3 p.m. PRESSURE } (\pm 0.82).$$

Its r^2 value was thirteen per cent. In Devonport the sexes were very similar in their predictor variables:

$$\text{DB} = -6.2 + 0.546 \text{ 3 p.m. COOLING} - \\ 0.12 \text{ MAXIMUM TEMPERATURE (64DB)} \\ (\pm 0.91)$$

$$\text{DG} = -54 + 0.43 \text{ 3 p.m. COOLING} + 0.12 \\ \text{9 p.m. COOLING} - 0.09 \text{ MAXIMUM TEMPERATURE} \\ (64\text{DB}) (\pm 0.87).$$

Their r^2 values were nineteen and seventeen per cent respectively. Like Hobart girls, both sexes' wheeziness was related to the atmospheric cooling associated with frontal activity but was affected by the maximum temperatures of two months ago. This probably described some form of acclimatisation. In summary, only three of the regressions gave reasonable proportions of explanation. Most relationships seemed to be related to the atmospheric cooling occurring during the passage of cold fronts. This reaction was particularly strong in the coastal areas. In the Midlands, however, the female morbidity was strongly related to high afternoon pressure.

Anticyclone to the North of Tasmania

In this synoptic situation, Hobart males' morbidity gained the highest explanation found in any group, eighty-four per cent, where:

$$\begin{aligned} \text{HB} = & 171.2 + 0.026 \text{ 3 p.m. RELATIVE HUMIDITY} + \\ & 0.92 \text{ evaporation} + 0.11 \text{ 9 p.m. RELATIVE} \\ & \text{HUMIDITY} - 0.176 \text{ 3 p.m. PRESSURE} - \\ & 0.092 \text{ 9 p.m. WINDSPEED} + 0.387 \text{ RAIN} - \\ & 0.21 \text{ 9 a.m. COOLING (26 DB)} (\pm 0.45). \end{aligned}$$

Female morbidity was not so clearly defined; its r^2 value was only twenty-one per cent:

$$\begin{aligned} \text{HG} = & 4.55 - 0.29 \text{ MINIMUM TEMPERATURE (26DB)} \\ & (\pm 2.13). \end{aligned}$$

In the Midlands too, the male morbidity was better explained than the female with r^2 values of fifty-nine per cent and sixteen per cent respectively:

$$\begin{aligned} MB = & -93.69 + 0.091 \text{ 3 p.m. PRESSURE} + \\ & 0.084 \text{ 3 p.m. COOLING} + 0.075 \text{ DIURNAL} \\ & \text{RANGE } (\pm 0.42) \end{aligned}$$

$$MG = 0.638 = 0.04 \text{ DIURNAL RANGE } (\pm 0.33).$$

Significant regressions were impossible to calculate for Burnie females or Devonport males but reasonable equations were obtained for Burnie boys and Devonport girls:

$$\begin{aligned} BB = & -0.18 - 0.14 \text{ MINIMUM TEMPERATURE (66DB)} \\ & + 0.145 \text{ 9 a.m. DRY-BULB TEMPERATURE } (\pm 0.72) \end{aligned}$$

$$\begin{aligned} DG = & 32.36 + 0.33 \text{ 9 a.m. DRY-BULB TEMPERATURE} \\ & + 0.1 \text{ MAXIMUM TEMPERATURE (64DB)} - \\ & -0.34 \text{ 3 p.m. WET-BULB TEMPERATURE} + \\ & 0.172 \text{ 3 p.m. PRESSURE} - 0.202 \text{ 9 p.m.} \\ & \text{PRESSURE} - 0.168 \text{ 3 p.m. COOLING } (\pm 0.86) \end{aligned}$$

Their r^2 values were forty-eight and fifty-nine per cent.

The relationships described by the equations were not so easy to discern as those in the previous class. This was especially true of the Hobart male morbidity equation. The many predictor variables of opposite sign that additively explained eighty-four per cent of the variation in morbidity seemed unrelated to each other. The first variable in the regression usually has the highest level of explanation and was therefore most important. Relative humidity measured at 3 p.m. in the afternoon was significantly related to Hobart boys' wheezing under these conditions as was rain. Sometimes when an anticyclone had been to the North it was coupled with a cyclone to the South and Hobart could have been affected more

than any other location by the ensuing atmospheric mixing and its subsequent cloud cover, high humidities and rainfall. Further North, the Midlands groups were responding to 3 p.m. pressure, 3 p.m. cooling and diurnal range. Yet further North, high 9 a.m. dry-bulb temperatures were of importance in Burnie and Devonport. It would seem that the heat of the air advected from the mainland of Australia affected these Northern Tasmanian asthmatics. In the Midlands the clear skies and continental air probably resulted in increased radiation cooling at night and a consequently increased diurnal range. Hobart, meanwhile, was affected by a different type of system at the boundary of the Northern anticyclone's extent.

Anticyclone over Tasmania

This situation was four times as common as that of an anticyclone to the North. Because of this, it was probably of greater heterogeneity. This, in part, may have accounted for the lower levels of explanation achieved. Again, Hobart males gave the highest r^2 value, forty-one per cent. Only half of the groups gained over twenty per cent explanation of their wheezing from weather variables. The regression equations for Hobart were:

$$\begin{aligned} \text{HB} = & -169.75 + 0.162 \text{ 9 p.m. PRESSURE} + 0.059 \text{ GUST} \\ & \text{SPEED} + 0.055 \text{ 9 a.m. RELATIVE HUMIDITY} \\ & + 0.185 \text{ DIURNAL RANGE} - 0.066 \text{ MAXIMUM} \\ & \text{TEMPERATURE (11DB)} (\pm 1.33) \end{aligned}$$

$$\begin{aligned} \text{HG} = & 5.23 - 0.198 \text{ 9 a.m. WET-BULB TEMPERATURE} \\ & - 0.091 \text{ MAXIMUM TEMPERATURE (11DB)} + 0.567 \\ & \text{RAIN} (\pm 1.33) \end{aligned}$$

Their r^2 values were forty-one and twenty-seven per cent.

The sexes' wheezing seemed to be related to different aspects of this weather pattern. Such differences provided supportive evidence for aetiological divergence between male and female morbidity. Boys' morbidity was related to the increased range in temperature associated with high pressure and the girls' morbidity was related to the reduced temperature associated with rainfall.

In the Midlands and on the Northwest Coast the levels of explanation were fairly low:

$$\begin{aligned} \text{MB} = & 1.97 - 0.093 \text{ MINIMUM TEMPERATURE (43DB)} - 0.062 \\ & \text{MAXIMUM TEMPERATURE (9DB)} + 0.064 \text{ MINIMUM} \\ & \text{TEMPERATURE (3DB)} (\pm 1.0) \end{aligned}$$

$$\begin{aligned} \text{MG} = & 0.83 = 0.095 \text{ 3 p.m. WET-BULB TEMPERATURE} + 0.079 \\ & \text{9 a.m. WET-BULB TEMPERATURE} (\pm 0.67) \end{aligned}$$

$$\text{BB} = 3.48 - 0.139 \text{ MAXIMUM TEMPERATURE (74DB)} (\pm 1.49)$$

$$\begin{aligned} \text{BG} = & -0.58 + 0.091 \text{ 9 a.m. WET-BULB TEMPERATURE} - 0.038 \\ & \text{9 a.m. WINDSPEED} (\pm 0.55) \end{aligned}$$

$$\text{DB} = 1.61 - 0.09 \text{ DIURNAL RANGE} (\pm 1.13)$$

$$\begin{aligned} \text{DG} = & 0.19 + 0.17 \text{ MINIMUM TEMPERATURE (17DB)} - 0.1 \\ & \text{9 a.m. WET-BULB TEMPERATURE} (\pm 1.1). \end{aligned}$$

Their r^2 values were 21, 8, 15, 25, 7 and 19 per cent respectively. Most of the relationships were with the minimum and maximum and wet-bulb temperatures. Burnie girls' morbidity was the most interesting of this group because of the combination of high

morning wet-bulb temperature and low windspeed. Hot, dry and calm conditions would have been expected to exacerbate pollution levels in this location.

Persistent Anticyclone Over Tasmania

These data described days on which Tasmania had been dominated by anticyclonic conditions for at least three days. In such conditions inversions were more likely to have occurred; trapping pollutants and concentrating their ambient levels. Compared to the last category, of which this is a subset, the explanatory power of the regressions was generally enhanced. The Hobart equations were of this form:

$$\begin{aligned} \text{HB} = & -147.8 + 0.14 \text{ 3 p.m. COOLING (8DB)} + 0.14 \text{ 9 a.m.} \\ & \text{PRESSURE} + 0.2 \text{ 9 a.m. WINDSPEED} - 0.05 \text{ AVERAGE} \\ & \text{WINDSPEED} + 0.34 \text{ 3 p.m. COOLING (1DB)} + 0.15 \\ & \text{MINIMUM TEMPERATURE (26 DB)} (\pm 1.0) \end{aligned}$$

$$\begin{aligned} \text{HG} = & 10.62 - 0.5 \text{ MINIMUM TEMPERATURE} - 0.53 \text{ 9 a.m.} \\ & \text{COOLING (26DB)} (\pm 0.84). \end{aligned}$$

These equations accounted for sixty-nine and sixty per cent of the variation in the boys and girls wheezing respectively. The major effect was due to atmospheric cooling either by radiation at night or by sea breezes in the afternoon.

In the Midlands, each equation had a single weather component:

$$\text{MB} = -1.6 + 0.13 \text{ 9 a.m. COOLING } (\pm 0.64)$$

$$\text{MG} = 64.5 - 0.063 \text{ 3 p.m. PRESSURE } (\pm 0.78).$$

The explanation here was much lower, twenty-four per cent of male wheezing and fourteen per cent of female wheezing. The male wheezing was, like Hobart, linked to atmospheric cooling; cool mornings especially. Female wheezing in these conditions, similar to female wheezing when anticyclones were stationed over the Bight, was again related to afternoon pressure.

In Burnie, male wheezing was related to low maximum temperatures ten weeks previously and to high 9 a.m. dry-bulb temperatures and to low minimum temperatures.

$$\begin{aligned} \text{BB} = & -3.57 - 0.19 \text{ MAXIMUM TEMPERATURE (74DB)} + 0.77 \\ & 9 \text{ a.m. DRY-BULB TEMPERATURE} - 0.32 \text{ MINIMUM} \\ & \text{TEMPERATURE } (\pm 1.12). \end{aligned}$$

This combination of variables gave an r^2 value of sixty per cent. For females in Burnie only half of this amount was explained.

$$\begin{aligned} \text{BG} = & 0.45 - 0.04 \text{ 3 p.m. WET-BULB TEMPERATURE} + 0.2 \\ & 9 \text{ a.m. WET-BULB TEMPERATURE} - 0.16 \text{ 9 a.m. DRY-BULB} \\ & \text{TEMPERATURE } (\pm 0.36). \end{aligned}$$

The association with low 3 p.m. wet-bulb and high 9 a.m. wet-bulb temperatures linked with the pollution scenario described in section four, figure 4.4.2. This is a situation of inversion potential acting in combination with an afternoon sea-breeze to increase fumigation from factory effluent.

It was not possible to obtain a statistically significant regression for Devonport female morbidity but the male equation was as follows:

$$\begin{aligned} \text{DB} = & 5.34 - 0.17 \text{ MAXIMUM TEMPERATURE} - 0.09 \text{ 3 p.m.} \\ & \text{WET-BULB TEMPERATURE } (\pm 1.147) \end{aligned}$$

This gave an r^2 value of thirty per cent. Morbidity was obviously related to days of low maximum temperatures and 3 p.m. wet-bulb temperature probably due to a sea-breeze affect.

Cyclone over the Great Australian Bight

Under this synoptic condition, no significant regressions could be calculated for either Hobart or Midlands wheezing. This situation usually results in the advection of continental air from the North across the Bass Strait to the North Coast of Tasmania. This may explain the localised nature of the effect and the strong relationships with temperature. The regressions for the Northwest-Coast dwellers were:

$$BB = 2 - 0.13 \text{ MINIMUM TEMPERATURE (27DB)} (\pm 0.86)$$

$$\begin{aligned} BG = & 1.46 + 0.05 \text{ MINIMUM TEMPERATURE (27DB)} - 0.15 \\ & \text{DIURNAL RANGE} - 0.09 \text{ MAXIMUM TEMPERATURE (74DB)} \\ & + 0.04 \text{ MAXIMUM TEMPERATURE (35DB)} + 0.013 \text{ RAIN} \\ & (\pm 0.13) \end{aligned}$$

$$DB = 2 - 0.14 \text{ MINIMUM TEMPERATURE (17DB)} (\pm 0.7)$$

$$DG = -94.4 + 0.99 \text{ 3 p.m. PRESSURE} (\pm 1.2)$$

These explained 23, 75, 37 and 23 per cent of their respective morbidity variations. Burnie girls' wheezing was most effectively modelled with seventy-five per cent explanation. This high level was only achieved, however, by using the three lagged variables. Both Burnie morbidity regressions possessed minimum temperature lagged twenty-seven days as the major component.

Cyclone over Tasmania

This weather type gave good fits between weather and wheezing with only two exceptions, Burnie girls and Midlands girls. In Hobart, males and females gave r^2 values of seventy-nine and eighty-four per cent:

$$\begin{aligned} \text{HB} = & 6.58 - 0.022 \text{ AVERAGE WIND SPEED} + 0.15 \text{ 9 a.m.} \\ & \text{WIND SPEED} - 0.19 \text{ MAXIMUM TEMPERATURE (11DB)} \\ & - 0.12 \text{ 3 p.m. WIND SPEED } (\pm 0.48) \end{aligned}$$

$$\begin{aligned} \text{HG} = & 11.92 - 0.5 \text{ MINIMUM TEMPERATURE (26DB)} - 0.49 \\ & \text{9 a.m. COOLING (26DB)} + 0.01 \text{ AVERAGE WIND SPEED} \\ & - 0.2 \text{ DIURNAL RANGE } (\pm 0.57). \end{aligned}$$

In the Midlands the boys' regression accounted for ninety-two per cent of the wheezy variation:

$$\begin{aligned} \text{MB} = & -84.4 + 0.08 \text{ 3 p.m. PRESSURE} + 0.07 \text{ MINIMUM} \\ & \text{TEMPERATURE (2DB)} + 0.07 \text{ MAXIMUM TEMPERATURE} \\ & + 0.07 \text{ 9 a.m. COOLING } (\pm 0.22). \end{aligned}$$

This was bettered by Burnie boys whose equation had an r^2 value of ninety-seven per cent:

$$\begin{aligned} \text{BB} = & 119.5 - 0.43 \text{ 9 a.m. DRY-BULB TEMPERATURE} + 0.17 \\ & \text{MINIMUM TEMPERATURE (66DB)} - 0.25 \text{ MAXIMUM} \\ & \text{TEMPERATURE (74DB)} - 0.11 \text{ PRESSURE} - 0.21 \text{ 3 p.m.} \\ & \text{WIND SPEED} + 0.44 \text{ MINIMUM TEMPERATURE} - 0.04 \text{ RAIN} \\ & - 0.025 \text{ 9 a.m. WIND SPEED } (\pm 0.16). \end{aligned}$$

The Burnie girls' regression was insignificant and the Devonport girls' equation gave an r^2 value of forty-five per cent.

Devonport boys' multiple regression explained eighty-eight per cent of their morbidity variations:

$$\begin{aligned} \text{DB} = & 11.9 - 0.04 \text{ DIURNAL RANGE} + 0.1 \text{ RAIN} - 0.42 \\ & \text{3 p.m. COOLING} - 0.36 \text{ 3 p.m. WIND SPEED} - 0.3 \\ & \text{9 a.m. DRY-BULB TEMPERATURE } (\pm 0.62). \end{aligned}$$

$$DG = 1.82 - 0.13 \text{ MAXIMUM TEMPERATURE} + 0.15 \text{ MINIMUM TEMPERATURE (27DB)} (\pm 1.11).$$

Generally, this type of weather pattern seemed to affect more groups more strongly than any other type considered hitherto, and would be expected to provide strong measures for successful modelling. Wind speeds and extremes of temperature played the dominant roles in these relationships.

Cut-Off Cyclones

These cyclonic conditions, although responsible for strong weather changes, did not produce as striking explanatory models as did the orthodox cyclones that affected Tasmania's weather. Insignificant regressions were obtained for girls from Hobart and Burnie. Hobart males' wheezing was described by a seven-variable model:

$$\begin{aligned} HB = & 63.6 + 0.35 \text{ MAXIMUM TEMPERATURE (11DB)} - 0.09 \\ & 9 \text{ a.m. PRESSURE} + 0.07 \text{ 9 a.m. RELATIVE HUMIDITY} \\ & + 1.72 \text{ 9 a.m. COOLING (26DB)} + 0.43 \text{ MINIMUM} \\ & \text{TEMPERATURE (26DB)} - 0.13 \text{ 3 p.m. COOLING (8DB)} \\ & - 0.07 \text{ 3 p.m. WIND SPEED } (\pm 0.62). \end{aligned}$$

This gave an r^2 value of sixty-two per cent. Meteorotropic response by this population to a cut-off cyclone was affected by four lagged variables ranging from one week to one month before.

In the Midlands, all the variables selected by the regressions were current ones:

$$\begin{aligned} MB &= -3.85 + 0.16 \text{ 3 p.m. COOLING} + 0.24 \text{ 9 a.m.} \\ &\text{ DRY BULB TEMPERATURE} = 0.06 \text{ 3 p.m. WIND SPEED} \\ &\quad - 0.09 \text{ MINIMUM TEMPERATURE } (\pm 0.46) \end{aligned}$$

$$MG = -2.03 + 0.17 \text{ 9 a.m. COOLING } (\pm 1.1).$$

These gave r^2 values of sixty-six and twenty-nine per cent.
For the Northwest Coast populations the equations were:

$$\begin{aligned} BB &= 2.87 - 0.14 \text{ MAXIMUM TEMPERATURE (74DB)} + 0.1 \\ &\quad \text{9 a.m. COOLING} - 0.11 \text{ MINIMUM TEMPERATURE (27DB)} \\ &\quad (\pm 0.66) \end{aligned}$$

$$\begin{aligned} DB &= 0.24 - 0.07 \text{ 3 p.m. DRY BULB TEMPERATURE} + 0.14 \\ &\quad \text{MINIMUM TEMPERATURE (27DB)} (\pm 0.81) \end{aligned}$$

$$\begin{aligned} DG &= -3.6 + 0.17 \text{ 9 a.m. COOLING} - 0.17 \text{ RAIN} + 0.13 \\ &\quad \text{MAXIMUM TEMPERATURE (46DB)} (\pm 0.92). \end{aligned}$$

Their respective r^2 values were 73, 42 and 54 per cent.

Stormy Westerlies

This extreme weather type managed to surpass the "cyclone stationed over Tasmania" pattern in regard to the greatest explanatory regression equation. The synoptic type was characterised by close isobars, high wind speeds and multiple cold fronts in quick succession. The Hobart equations were given by:

$$\begin{aligned} HB &= 144.4 + 0.04 \text{ MAXIMUM TEMPERATURE (2DB)} - 0.14 \\ &\quad \text{3 p.m. PRESSURE} - 0.02 \text{ AVERAGE WIND SPEED} - 0.08 \\ &\quad \text{3 p.m. WIND SPEED} - 0.03 \text{ 9 a.m. RELATIVE HUMIDITY} \\ &\quad - 0.34 \text{ EVAPORATION } (\pm 0.42) \end{aligned}$$

$$\begin{aligned} HG &= 264.9 - 0.32 \text{ 3 p.m. WIND SPEED} - 0.05 \text{ AVERAGE} \\ &\quad \text{WIND SPEED} - 0.17 \text{ 9 a.m. RELATIVE HUMIDITY} - 0.92 \\ &\quad \text{9 a.m. WET BULB TEMPERATURE} - 0.24 \text{ 9 a.m. PRESSURE} \\ &\quad + 0.55 \text{ DIURNAL RANGE} + 0.35 \text{ MAXIMUM TEMPERATURE} \\ &\quad \text{(2DB)} (\pm 1.6) \end{aligned}$$

These explained eighty-six and sixty-three per cent of the wheezing variation for males and females respectively.

The Midlands' regressions were both very successful. The male r^2 value was ninety-eight per cent and the female r^2 value was eighty-three per cent. The equations were as follow:

$$\begin{aligned} MB = & 57.74 - 0.27 \text{ MAXIMUM TEMPERATURE (9DB)} + 0.09 \\ & 3 \text{ p.m. WIND SPEED} + 0.31 \text{ MINIMUM TEMPERATURE} \\ & (3DB) - 0.304 \text{ MAXIMUM TEMPERATURE} + 0.21 \text{ RAIN} \\ & - 0.07 3 \text{ p.m. PRESSURE } (\pm 0.2) \end{aligned}$$

$$\begin{aligned} MG = & 5.7 - 0.33 \text{ MAXIMUM TEMPERATURE} + 0.23 \text{ MINIMUM} \\ & \text{TEMPERATURE (43DB)} - 0.11 \text{ MINIMUM TEMPERATURE} \\ & (2DB) (\pm 0.5). \end{aligned}$$

The Midlands boys' regression was matched by the Burnie boys' equation with an r^2 value of ninety-eight per cent. Burnie girls morbidity could not be fitted by the available variables to give a significant model. The Northwest Coast equations were structured thus:

$$\begin{aligned} BB = & -60.1 + 0.75 3 \text{ p.m. COOLING} + 0.43 \text{ MINIMUM TEMPERATURE} \\ & (27DB) + 0.104 3 \text{ p.m. PRESSURE} - 0.06 9 \text{ p.m. PRESSURE} \\ & (\pm 0.16) \end{aligned}$$

$$DB = 7.3 - 0.4 \text{ MAXIMUM TEMPERATURE } (\pm 0.64)$$

$$\begin{aligned} DG = & 4.05 - 0.82 \text{ MAXIMUM TEMPERATURE} + 0.7 \text{ MAXIMUM} \\ & \text{TEMPERATURE (46DB)} (\pm 0.96). \end{aligned}$$

The proportion of variation explained by each of these equations was 99, 69 and 81 per cent respectively.

Zonal Westerlies

This synoptic type was one of the most common situations found to affect Tasmanian weather. After the high level of explanation attained by regressions of wheezing and weather variables in cyclonic and stormy-westerly conditions, the r^2 values for the regressions under this weather pattern were disappointing. In the Midlands no significant regression equations could be calculated. The Hobart equations were:

$$HB = -0.21 + 0.44 \text{ 9 a.m. COOLING} - 0.01 \text{ AVERAGE WIND SPEED} - 0.09 \text{ MAXIMUM TEMPERATURE (11DB)} (\pm 1.3)$$

$$HG = 3.2 - 0.25 \text{ MINIMUM TEMPERATURE (26DB)} + 0.15 \text{ 3 p.m. COOLING (1DB)} - 0.1 \text{ 9 a.m. WIND SPEED} (\pm 1.5).$$

Their r^2 values were forty-two and thirty-seven per cent.

On the Northwest Coast the equations had smaller explanatory values. They were as follow:

$$BB = 4.05 - 0.18 \text{ MAXIMUM TEMPERATURE (74DB)} (\pm 1.1)$$

$$BG = -0.63 + 0.06 \text{ MINIMUM TEMPERATURE} - 0.06 \text{ MINIMUM TEMPERATURE (66DB)} + 0.05 \text{ MAXIMUM TEMPERATURE (35DB)} (\pm 0.54)$$

$$DB = 2.26 - 0.13 \text{ MAXIMUM TEMPERATURE} + 0.08 \text{ MINIMUM TEMPERATURE (27DB)} (\pm 1.0)$$

$$DG = 0.56 - 0.08 \text{ 9 a.m. WIND SPEED} + 0.11 \text{ MINIMUM TEMPERATURE (17DB)} (\pm 1.26).$$

In these cases, the r^2 values were 29, 22, 24 and 14 per cent.

All of the r^2 values for the eighty separate regression analyses are summarised in Table 5.3.1. It is obvious that in any synoptic situation the different populations demonstrate an individual response to the weather complex. In some areas, for certain synoptic types, wheezing can be almost totally explained by the additive effects of weather elements. In other areas, under different conditions, wheeziness is almost totally unrelated to weather. In the majority of situations, however, some portion of the variation in morbidity is explained by weather variables. The question now posed is ... "What relevance do these relationships have with future events?" One of the purposes of regression analysis is to be able to predict the value of the dependent variable given the several independent variables. Seven additional months of daily morbidity and weather data were collected specifically to answer this question.

Testing the Efficiency of the Regression Models

Regressions fitted to empirical data can be constructed to explain an optimum amount of variation in that data set. Often, however, the predictive equations lack efficacy when tested upon a different sample. The regression analyses completed in this section vary in their explanation of wheezing based upon the weather. How useful are these equations in describing morbidity levels in other populations than the ones they were based upon? From June 1st, 1978 to December 31st, 1978, morbidity and weather data collection was continued to acquire the necessary data upon which to test the models.

TABLE 5.3.1

Values of $r^2 \times 100$ for the regressions based on synoptic types

	HB	HG	MB	MG	BB	BG	DB	DG
Anticyclone over Bight	26	48	6	56	13	NS	19	17
Anticyclone to North	84	21	59	16	48	NS	NS	59
Anticyclone over Tasmania	41	27	21	8	15	25	7	19
Anticyclone over Tasmania (≥ 3 days)	69	60	24	14	60	31	30	NS
Anticyclone over Tasman Sea	51	20	70	NS	32	NS	NS	NS
Cyclone over Bight	NS	NS	NS	6	23	75	37	23
Cyclone over Tasmania	79	84	92	NS	97	NS	88	45
Cut-off Cyclone	62	NS	66	29	73	NS	42	54
Stormy Westerly	86	63	98	83	99	NS	69	81
Zonal Westerly	42	37	NS	NS	29	22	24	14

HB Hobart Boys

HG Hobart Girls

MB Midland Boys

MG Midland Girls

BB Burnie Boys

BG Burnie Girls

DB Devonport Boys

DG Devonport Girls

NS (No combination of variables could be obtained to yield an equation whose F-ratio reached significance at $p \leq 0.05$).

Given a series of observed morbidity values the intention was to generate a series of predicted values using the regression equations and then to examine how closely the expected and predicted series of values corresponded. This process was accomplished by taking the observed and expected values for each area-sex-synoptic classification and calculating the simple correlation coefficient between them. When this value was squared it gave r^2 values. These are tabulated in Table 5.3.2.

From the table it was observed that many of the low explanation models decreased further in explanatory power when tested on the new data. Most of the higher explanation models, however, decreased only slightly or actually increased in power when predicting the new values. With the exception of Burnie girls, every population demonstrated significant meteorotropic response to Stormy westerlies. The next most important synoptic situation was when a cyclone was stationed over Tasmania; here, five out of eight populations possessed more than seventy-five per cent explanation of their wheeziness. In situations of cut-off cyclones all the male populations exhibited strong meteorotropic behaviour and the females none, again stressing the regular finding of inter-sex difference. The only groups to respond at this level to persistent anticyclones were the Hobart sample and Burnie males. This obviously described some urban influence on morbidity linked to weather conditions. Hobart girls and Midlands girls' wheezing had strong relationships

TABLE 5.3.2

Values of $r^2 \times 100$ for the correlation between the observed and predicted levels of morbidity for June 1 - December 31, 1978 based on the several synoptic regressions

	HB	HG	MB	MG	BB	BG	DB	DG
Anticyclone over Bight	11	55	4	60	7	NS	5	8
Anticyclone to North	81	10	62	9	45	NS	NS	45
Anticyclone over Tasmania	35	12	9	2	6	12	2	3
Anticyclone over Tasmania (> 3 days)	75	57	10	6	65	31	19	NS
Anticyclone over Tasman Sea	32	5	63	NS	13	NS	NS	NS
Cyclone over Bight	NS	NS	NS	3	9	70	29	11
Cyclone over Tasmania	75	79	85	NS	87	NS	81	21
Cut-off Cyclone	58	NS	57	11	65	NS	51	19
Stormy Westerly	82	53	96	67	92	NS	72	75
Zonal Westerly	40	31	NS	NS	12	3	27	4

HB Hobart Boys

HG Hobart Girls

MB Midland Boys

MG Midland Girls

BB Burnie Boys

BG Burnie Girls

DB Devonport Boys

DG Devonport Girls

NS (No combination of variables could be obtained to yield an equation whose F-ratio reached significance at $p \leq 0.05$).

with anticyclones that were stationed over the Great Australian Bight and Hobart boys and Midlands boys demonstrated a meteorotropic response when anticyclones were to the North of Tasmania. Midlands boys, when an anticyclone was over the Tasman Sea, exhibited another meteorotropic relationship. When a cyclone was stationed over the Great Australian Bight only Burnie girls possessed a significant meteorotropic relationship with the weather.

The regressions given are all significant statistically and contribute something toward the explanation of morbidity. The separate efficiencies of each synoptic classification have been given in table 5.3.2 in the form of the percentage of explanation described by their r^2 values. These levels, however, do not reflect the average predictability of wheeziness in the several groups. To obtain the average level of explanation the entire year of observed values is correlated with the same time series, the constituent daily parts of which have been estimated by the appropriate synoptic regressions for those days. This process is repeated for the seven months of test data. The correlations between the observed and expected series are then squared to achieve a measure of explanation. These percentages form the first two columns of table 5.3.3.

In table 5.3.1 are displayed the explanatory efficiencies of the individual synoptic regressions using the original twelve months' data. In table 5.3.2 the explanatory efficiencies of the same regressions are illustrated when used upon the seven months of novel data. In table 5.3.3 the combined explanatory power

of the several synoptic regressions taken together are given for both the original year of data and the further seven months of data.

For comparative purposes and to emphasise the benefits obtained from the synoptic analyses, regressions were also calculated for the entire data series without regard for these synoptic patterns. One of these 'annual' equations was given at the beginning of this section for Hobart girls; it attained twenty-eight per cent explanations. The third column in table 5.3.3 gives the r^2 values for each of the 'annual' regressions based on the twelve months' data. These equations were also tested on the new data by calculating the correlation between the observed and expected values. The squares of these correlations are given in the fourth column of table 5.3.3 as percentages.

It is seen from the table that the synoptic regressions together give the best average explanation of morbidity fluctuations. The level of explanation based on the test data is, however, lower than that for the base period. This is to be expected. In all cases the Hobart samples gained the greatest average amount of explanation. In the test period the Northwest Coast regressions lose their efficacy. The regressions based on the twelve months' data without regard to synoptic patterns generally achieve very low levels of explanation. A comparison of the r^2 values with and without vindicates the use of separate models for the different synoptic conditions.

TABLE 5.3.3

Values of $r^2 \times 100$ based on the correlation between observed and expected levels of morbidity

	<u>SYNOPTIC REGRESSION</u>		<u>ANNUAL REGRESSION</u>	
	Base Period	Test Period	Base Period	Test Period
Hobart Boys	65	54	21	7
Hobart Girls	50	42	28	9
Midlands Boys	38	29	11	4
Midlands Girls	32	27	13	4
Burnie Boys	45	30	14	3
Burnie Girls	24	12	5	0
Devonport Boys	33	11	6	1
Devonport Girls	38	18	11	2

Influences of Pollution

The only indicator of atmospheric quality, other than the meteorological variables that was analysed to assess its relationship with wheezing was the level of acid-gases for Hobart only. (Ionisation is discussed in appendix 3). In order to maintain comparability between areas, this index of pollution was not included in the previous regression analyses. It was, however, included in several additional regressions that were specifically designed to examine its relationship to wheezing. The contribution it made to these analyses was very small. The maximum amount of variation in wheezing that it explained was in a regression for Hobart boys' wheezing in Summer. The equation was:

$$\text{HB (Summer)} = -63.6 + 0.064 \text{ 9 p.m. PRESSURE} + 0.016 \text{ GUST SPEED} - 0.039 \text{ POLLUTION } (\pm 1.03).$$

The r^2 value for the regression was twenty-five per cent of which the pollution index accounted for only 2.7 per cent.

Interestingly, its relationship to wheezing was negative.

In prophylactic terms and at the levels experienced in Hobart, the influence of acid-gas variation may be disregarded in terms of wheezing.

Summary

It has been shown that a reasonable, average prediction can be achieved if several synoptic-specific regression models are combined. However, is average-level forecasting the desired goal? Should not the prediction of distress be oriented toward the extreme and peak events? For a large part of the time in

many parts of the State, the incidence of wheezing is low; of what use is a predicted level at these times? Surely the interpretation of a low-explanation regression equation is that there is no relationship, or very little of one, between wheezing and weather in the given situation-sex-synoptic category. One can assume that the non-significant regressions mean that in their specific times populations and places, any wheezing that occurs is not related to weather variables. This could be due either to the lack of wheezing incidence during these periods or to the activity of other triggers. This study does not take into account the daily variation in spores, pollens, mites, particulates, infections, psychological trauma or hypochondriasis and added to the effects of all these agents is the unavoidable, random, stochastic error present in every study of human variation.

However, this research has been concerned with biometeorological affects upon wheezy breathing and in situations where there appears to be no "weather affect" more research is needed in other fields to clarify aetiological factors. Actually, from a biometeorological point of view, many of the variables included in the regression equations may not be directly affecting wheezing but may be acting indirectly by influencing, for example, spore levels or human activity patterns.

Whatever the mechanisms involved, an appropriate forecasting strategy would be to use the site-sex-synoptic specific regression equations to predict levels of wheeziness for particular situations on a day-to-day basis. On many

occasions, for example in Hobart when a cyclone is stationed over the Great Australian Bight or in the Midlands during zonal westerlies, no concern would be necessary. Parents of children or the sufferers themselves can be educated, on a 'rule of thumb basis', to recognise the 'risky' synoptic patterns given in weather announcements on television or in the newspaper. In times of high potential risk preventive measures can be taken. These might take the form of drug therapy, extra clothing or confinement to the house. It would also be feasible to build the calculation of risk into the meteorological bureau reports. This could then be included in the televised broadcasts for very little expense. Discomfort indices are well known examples of this sort of application in North America.¹

The avoidance of meteorological stress is probably advisable only for the major hazards. Climatotherapists argue that too much protection may increase the effect of milder stresses. This is again related to the concept of 'morbidity limit'. Small stresses build up the asthmatic's resistance to stronger events. In situations of over-protection this threshold can diminish to the extent that an asthmatic's risk is actually increased. In order to avoid the damage that can be caused by severe attacks of dyspnoea a reasonable course of primary prevention would be to combine normal life-style objectives with increased precautions during high-risk periods.

5.4 Study Limitations and Recommendations for Future Research

The limitations of this research fell into three categories: human, environmental and design problems. These were not mutually exclusive. After the analysis of the 1968 survey it was decided to limit the monitoring of asthmatics-wheezy breathers to four major defined concentrations, to those who lived in two towns on the Northwest Coast, in the Midlands and in Hobart. This was necessary because of the limited resources available; a state sample would have been unmanageable. This decision virtually limited the study to the city of Hobart and the industrial towns of the Northwest Coast for the population numbers in the Midlands were small and diffuse. The next limitation was in the selection of wheezers. School Health Services provided the best sample but as always with the identification of morbid populations, unknowable biases were probably involved in the detection of wheezers by these means. In addition, greater numbers would have been desirable, especially in the Midlands, so that the wheezy records could have been subdivided not only by sex and residence but also by clinical types. In this regard, the absence of any allergy information was disappointing. Nor could any reasonable avenue be established to remedy this known deficiency. Sample numbers could have been increased by enlarging the age-range but this was unacceptable because of the probable increases in trend, variance and error.

The environmental data were also limited. Nowhere in Tasmania was atmospheric quality being monitored on a continuous basis with the exception of the C.S.I.R.O. [Australian Commonwealth Scientific and Industrial Organisation] baseline monitoring unit at Cape Grim in the far Northwest where no population relevance was achieved. The range and maybe even the quality of meteorological data also varied enormously from station to station within the State. Pollen and spore counts would have been other valuable inputs had they been available. Earlier work on these facets in Tasmania, however, had proved to be inconclusive.³

Because of inevitable limitations of personnel, equipment and time the findings published in this thesis are limited to the specific geographic areas that were examined (Launceston and the East and West Coasts are the major areas not covered). Although the regression equations are limited to age-sex-site specific groups, the method is applicable to any other closely defined group. In fact, an appropriate direction for further research would be to replicate similar studies for older cohort samples and for distant geographic and climatic regions. Given that meteorotropic mechanisms are observed to vary within this small island state much could be gained from comparisons at a national scale. The results of this research pose new questions that require answers. Why do the sexes' morbidity indices differ in their geographic distribution and meteorotropic response? Do biometeorological relationships with wheeziness change with

age? Are some of the differences due to unknown biases in the populations that were studied?

The analyses in section two showed that a population of childhood wheezers was, in many ways, not homogeneous. Unfortunately, because of the small numbers available, this point had to be ignored when gathering the samples from the 1971 birth cohort for prospective study. Although all these children possessed the phenomenon of wheezy breathing in common, differences in allergic status, nutrition, socioeconomic class etc. may have produced differences in the recorded history of episodes during the study period.

Answers to these questions would, obviously, be best achieved by a multi-disciplinary team effort. To gain further insights into environmental pathogenesis or biometeorological mechanisms would require a much larger sample of far greater geographical coverage. Given sufficient funding many of the limitations and imbalances described could be redressed or reduced. An ideal study would include the daily monitoring of several hundred members in each state of a nation-wide stratified random sample of the wheezy population and controls contemporaneously with a similarly based atmospheric quality surveillance network. With sufficient numbers, the wheezers could be separated into contrasting groups based on various characteristics such as allergy, age, sex and socioeconomic status for each of the regions. The minimum period of observation would be for one year but if cycles and trends were of interest it would be necessary to extend this period to at least five years. This scale of endeavour would be prohibitively expensive

and the results would probably be academic rather than useful.

A practicable alternative would be to repeat the biometeorological study described in section four on samples of 50-100 schoolchildren asthmatics in each of the capital cities of Australia. This could be carried out quite easily given the assistance of the local asthma foundations and the school health services. The monthly diary sheets could be given to parents as a booklet containing the entire year's record. Local asthma foundation personnel could telephone or call regularly to remind and encourage parents to record the episodes. Similar procedures could be used to collect the diaries at the end of the year. Correlation and regression analysis using data from local weather stations would result in national geographic/meteorological risk models of asthmatic distress.

The natural history of asthma and wheezy breathing remains largely unknown. In this thesis geographic and meteorological effects have been demonstrated and a reasonable method of predicting spatiotemporal risks to asthmatics has been presented. Much work remains to be done on the geographic analysis of future surveys of the 1961 Tasmanian birth cohort. One question, for example, that may be answered from these survey data is "what effect does changing residence have on prognosis?" This question is related to other environmental-geographic questions notably "what effect does long-term urban residence have on prognosis and spirometry?"

These and other queries can only be answered by the continued prospective surveillance of cohort populations. For this reason, the continuation of the Tasmanian Asthma Survey throughout the adult life of the 1961 birth cohort is strongly recommended. Also, the replication of this survey in other states of Australia and in other countries is to be encouraged for it is only by the use of standard methods⁴ that intra-national or international comparisons can be achieved. Research along these lines is currently being executed in Queensland⁵ in an attempt to verify the existence of the high asthma prevalence strip from Grafton to Granton that was described earlier in section one. If similar surveys were to be conducted in Sydney, Melbourne, Adelaide and Perth, the geographic factors linked to asthma prevalence could be established more firmly.

"For if we rightly understood the different temperatures of the Air and Winds, and how to apply this to many distempers, it might possibly prove the most successful part of Physick." 6

Section 5

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APPENDIX ONE

THE 1968 SURVEY OF THE 1961 TASMANIAN BIRTH COHORT

TASMANIAN ASTHMA SURVEY

CHILD'S MEDICAL HISTORY — CONFIDENTIAL

THIS SECTION IS FOR OFFICE USE ONLY

Code

.....Survey Number

CHILD'S SURNAME.....

CHILD'S CHRISTIAN NAME.....

SCHOOL HEALTH NUMBER.....
(not to be coded)

.....Sex.....

.....School Name.....

.....Child's School Number

TO BE FILLED IN BY PARENT, GUARDIAN OR RELATIVE

Important

(a) Before completing this form, please indicate your relationship to the child named in the panel above by putting a cross in the appropriate square below:

MOTHER

FATHER

STEP-PARENT OR
ADOPTED PARENT
OR GUARDIANOTHER
RELATIONNO
RELATION☐☐☐☐☐

(b) Has the child named above got a twin, or any brothers or sisters born in the same year (that is 1961)? (put a cross in the appropriate square)

YES

NO

☐☐

If the answer is YES, please give Christian names and the name of the school the brother or sister attends:

Name.....

School.....

(c) Please state the number of living full brothers and sisters in the family (excluding any born in 1961).

(d) If you do not read English well and have difficulty understanding the questions for this reason, please put a cross here ☐

(e) Please name the local doctor or hospital usually attended by this child for any ailment.

HOW TO ANSWER THE QUESTIONS:

In most cases, unless otherwise indicated, you are asked to answer YES or NO by putting a cross ☒ in the appropriate square. Sometimes, if the answer is not simply YES or NO, there are more than two squares provided, but you should still put a cross in only one of the several squares. Only three questions (relating to months of the year) may need a cross in more than one square, but these exceptions are specifically mentioned.

Please make sure that ALL questions are answered; please do not omit one, as this might mean we can not use the other information you will have given us. We understand that the answers to some questions can only be approximate, just as we understand that one's memory cannot always be perfectly accurate. If you wish to add more information or more details, please do so on a separate sheet of paper. Do NOT write extra notes on the form, and always answer the question with a cross in the most appropriate square, even if you add extra notes on a separate piece of paper.

Note that there are really four separate forms — the first deals with the health of the child named in the panel above, the second deals with the health of the mother, the third with the health of the father, and the fourth with the health of brothers and sisters. All these are equally important in helping us to understand the importance of the whole family in respiratory and allergic illnesses.

Office use only

1. (a) Where was he/she born?

Tasmania

Other Australian State

United Kingdom, New Zealand, South Africa, Canada,
United States of America

Other Overseas country

(Please Name.....)

☐
☐
☐
☐

(b) Date of birth.....

	YES	NO
2. Did he/she have infantile (baby) eczema?	<input type="checkbox"/>	<input type="checkbox"/>
3. Has he/she ever had eczema in the creases (bends) of elbows, wrists, or knees?	<input type="checkbox"/>	<input type="checkbox"/>
4. How was he/she fed in the first three months of life?		
Breast only	<input type="checkbox"/>	
Bottle only	<input type="checkbox"/>	
Breast and bottle	<input type="checkbox"/>	
5. Does he/she have a stutter or stammer?	YES <input type="checkbox"/>	NO <input type="checkbox"/>
6. Has he/she a habit of biting the fingernails?	<input type="checkbox"/>	<input type="checkbox"/>
7. During the first three years of life did you have to take the child to the doctor because of difficulties with feeding?		
Often	<input type="checkbox"/>	
Occasionally	<input type="checkbox"/>	
Never	<input type="checkbox"/>	
8. For how much time in the last twelve months has the child been confined to the house because of chest illnesses?		
Not at all	<input type="checkbox"/>	
One to seven days	<input type="checkbox"/>	
Eight to thirty days	<input type="checkbox"/>	
More than a month	<input type="checkbox"/>	
9. Has he/she had the tonsils removed?	YES <input type="checkbox"/>	NO <input type="checkbox"/>
10. Has he/she had more than two sore throats or attacks of tonsillitis in the past twelve months?	<input type="checkbox"/>	<input type="checkbox"/>
11. Have you ever been told by a doctor that he/she had pneumonia or pleurisy?		
No, Never	<input type="checkbox"/>	
Yes, Once or twice	<input type="checkbox"/>	
Yes, More than twice	<input type="checkbox"/>	
12. Have you been told by a doctor that he/she is allergic to any foods or medicines?	YES <input type="checkbox"/>	NO <input type="checkbox"/>
13. Does he/she get hives?		
Never	<input type="checkbox"/>	
Once or twice a year	<input type="checkbox"/>	
More than twice a year	<input type="checkbox"/>	
14. Has he/she at any time in his/her life suffered from attacks of asthma or of wheezy breathing?	YES <input type="checkbox"/>	NO <input type="checkbox"/>
(Note: Please regard "asthma" and "wheezy breathing" as being much the same thing for this survey; we do not ask you to try to tell the difference)		
The following questions (Nos. 15 to 21) relate to the details of these illnesses, and need to be answered if the answer to question 14 was YES. If the answer to question 14 was NO, omit these questions, and go on to question 21.		
15. How long is it since the last attack?		
Less than a month ago	<input type="checkbox"/>	
Over one but less than three months ago	<input type="checkbox"/>	
Over three but less than six months ago	<input type="checkbox"/>	
Over six but less than twelve months ago	<input type="checkbox"/>	
Over one year but less than two years ago	<input type="checkbox"/>	
Over two years ago	<input type="checkbox"/>	
16. On the average (as near as you can say), how often do these attacks tend to occur over the last two years or so?		
About once in twenty-four hours	<input type="checkbox"/>	
About once a week	<input type="checkbox"/>	
About once a fortnight	<input type="checkbox"/>	
About once a month	<input type="checkbox"/>	

17. On the average (as near as you can say), how long do these attacks usually last (with usual treatment)?

About once every three months
 About once every six months
 About once a year (or less often)
 No attacks at all in the last two years
 Less than twelve hours
 A day or so
 A week or so
 A month or so
 "Continuous" (never free of asthma or wheezing for more than a day or two)

18. At what age did these attacks begin?

Under 1 year
 Over 1 and under 2 years
 Over 2 and under 3 years
 Over 3 and under 4 years
 Over 4 and under 5 years
 Over 5 and under 6 years
 Over 6 and under 7 years
 Over 7 years

19. Since the attacks began, approximately how many has he/she had altogether

One attack only
 Two to five attacks
 Six to ten attacks
 Eleven to twenty attacks
 Over twenty attacks

20. If the attacks tend to be more frequent or more severe at any particular time of the year, indicate the "bad" months by putting a cross in the appropriate square(s) (but DO NOT put a cross against more than FOUR of the twelve months). If no month is worse than the others, leave all squares blank.

JAN. FEB. MCH. APRIL MAY JUNE JULY AUG. SEPT. OCT. NOV. DEC.

- 21. Has he/she at any time in his/her life suffered from attacks of bronchitis or attacks of cough with sputum (phlegm) in the chest ("loose" or "rattly" cough)?

(Note: Please regard "bronchitis" and "cough with sputum (phlegm) in the chest," and "loose or rattly cough" as being much the same thing for this survey; we do not ask you to try to tell the difference).

YES NO
☐ ☐

The following questions (Nos. 22-27) relate to the details of these illnesses, and need to be answered if the answer to question 21 was YES. If the answer to question 21 was NO, omit these questions and go on to question 28.

22. How long is it since the last attack?

Less than a month ago
 Over one but less than three months ago
 Over three but less than six months ago
 Over six but less than twelve months ago
 Over one year but less than two years ago
 Over two years ago

23. On the average (as near as you can say) how often do these attacks tend to occur over the last two years or so?

About once in twenty-four hours
 About once a week
 About once a fortnight
 About once a month
 About once every three months
 About once every six months
 About once a year (or less often)
 No attacks at all in the last two years

24. On the average (as near as you can say), how long do these attacks usually last (with usual treatment)?

Less than twelve hours
 A day or so
 A week or so
 A month or so
 "Continuous" (never free of loose cough for more than a day or two)

☐
☐
☐
☐
☐

25. At what age did these attacks begin?

Under one year
 Over 1 and under 2 years
 Over 2 and under 3 years
 Over 3 and under 4 years
 Over 4 and under 5 years
 Over 5 and under 6 years
 Over 6 and under 7 years
 Over 7 years

☐
☐
☐
☐
☐
☐
☐
☐

26. Since the attack began, approximately how many has he/she had altogether?

One attack only
 Two to five attacks
 Six to ten attacks
 Eleven to twenty attacks
 Over twenty attacks

☐
☐
☐
☐
☐

27. If the attacks tend to be more frequent or more severe at any particular time of the year, indicate the "bad" months by putting a cross in the appropriate square(s) (but DO NOT put a cross against more than FOUR of the twelve months). If no month is worse than the others, leave all squares blank.

JAN. FEB. MCH. APRIL MAY JUNE JULY AUG. SEPT. OCT. NOV. DEC.
☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐

- 28. Does he/she get attacks of "hay fever" (that is, sneezing, running or blocked nose, sometimes with itchy eyes or nose)?

YES NO
☐ ☐

If the answer to question 28 is NO, omit question 29.

29. If these hay fever attacks tend to be more frequent or more severe at any particular time of the year, indicate the "bad" months by putting a cross in the appropriate square(s) (but DO NOT put a cross against more than FOUR of the twelve months). If no month is worse than another, leave all squares blank.

JAN. FEB. MCH. APRIL MAY JUNE JULY AUG. SEPT. OCT. NOV. DEC.
☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐

YES NO
☐ ☐

30. Is he/she prone to "colds in the head" (that is, more than two or three colds a year)?

If the answer to question 30 is NO, omit question 31.

31. If these head colds tend to be more frequent or more severe at any particular time of the year, indicate the "bad" months by putting a cross in the appropriate square(s) (but DO NOT put a cross against more than FOUR of the twelve months). If no month is worse than another, leave all the squares blank.

JAN. FEB. MCH. APRIL MAY JUNE JULY AUG. SEPT. OCT. NOV. DEC.
☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐

YES NO
☐ ☐

32. Has he/she ever had a chest X-ray?

If the answer is YES:

Where was the X-ray taken?
 (Name of hospital, doctor or clinic)

What year was it taken? 19.....

Was it normal, as far as you know?

YES NO
☐ ☐

33. Has he/she ever attended a chest clinic as a contact of a person with tuberculosis?

☐ ☐

Please check your answers to all the questions: Thank you.

TASMANIAN ASTHMA SURVEY

CLINICAL DATA - CONFIDENTIAL

THIS SECTION FOR OFFICE USE ONLY

Code

..... Survey Number

CHILD'S SURNAME.....

CHILD'S CHRISTIAN NAME.....

SCHOOL HEALTH NUMBER.....
(not to be coded)

..... Sex.....

..... School Name.....

..... Child's School Number

Office use only

Date of Examination.....

Medical Officer's Number.....

Height (ins.).....

Weight (lbs.).....

Scales Calibration ☐ 28 lbs. =

56 lbs. =

Flexural Eczema (knees, elbows and wrists)

Generalised Eczema

Nasal Discharge

None

Present Mucoid

Present Coloured

Nasal Obstruction

Right

Left

Post Nasal Discharge

Chest Deformity

1. Kyphosis

2. Scoliosis

3. Funnel

4. Pigeon

5. "Asthmatic"

SEVERE	MILD	ABSENT

YES	NO
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

<input type="checkbox"/>
<input type="checkbox"/>
<input type="checkbox"/>

YES	NO
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

[OVER

Cough

Dry ☐Loose ☐Would not cough ☐

Audible Wheezing

YES ☐NO ☐

Auscultation

Normal ☐Rhonchi unilateral ☐bilateral ☐Rales unilateral ☐bilateral ☐

Ventilatory Function

Date of Test.....a.m./p.m.

Room Temperature.....°C

Spirometer Number.....

Co-operation

Satisfactory ☐Doubtful ☐Poor ☐YES ☐NO ☐

Spirometer Calibration checked

If Ventilatory Test not performed, state reason.....

Attach Spirogram

RESULTS (Office use only)

r.E.V. 0.5 (ml)

F.E.V. 1.0 (ml)

V.C. (ml)

Ratio F.E.V./V.C.

M.E.F.R. (ml/sec)

APPENDIX TWO

THE 1977 SURVEY OF THE 1971 TASMANIAN BIRTH COHORT

The questionnaires from appendix one were verbally administered to the children in the samples. None of the replies were pre-coded. In addition, four further questions were asked:

- a) what drugs or medicines do you use for your asthma?
- b) what drugs have you used in the past for your asthma?
- c) what do you think triggers your attacks of wheezing?
- d) can you remember where your worst attacks took place?

Some of the material is presented here in map or tabular form.

The wheezy morbidity series were collected by using a monthly diary sheet. An example of this and its accompanying instructions are included at the end of this appendix after the location maps. A location map was not included for the Midlands sample as the cases were virtually clustered at two points, Campbelltown and Ross. Both of these settlements were indicated on a map in section four (figure 4.3.1).

TABLE A.2.1
Average values of wheezy breathing by geographic area.

<u>BURNIE</u>				
	<u>Males</u>		<u>Females</u>	
	Wheezers	Remissions	Wheezers	Remissions
AGE OF ONSET*	1.5	0.8	1.8	2.3
TOTAL NUMBER**	17	10	10	7
DURATION***	4.5	1.5	1.4	1.5
<u>DEVONPORT</u>				
AGE OF ONSET	1.8	0.9	2.8	0.8
TOTAL NUMBER	35	23	30	7
DURATION	1.5	3.3	6.6	2.5
<u>MIDLANDS</u>				
AGE OF ONSET	0.5	3.0	0.8	-
TOTAL NUMBER	30	10	35	-
DURATION	10.6	4.5	4.0	-
<u>HOBART</u>				
AGE OF ONSET	1.5	0.8	1.0	1.6
TOTAL NUMBER	40	8.0	44	19
DURATION	1.9	2.5	3.9	1.4

* in years

** of attacks

*** in days

TABLE A.2.2

Average Weeks of chest illness experienced in last
twelve months by site

	BURNIE	DEVONPORT	MIDLANDS	HOBART
Male Wheezers	6.5	5.0	3.5	2.7
Male Remissions	2.0	3.0	0.3	1.4
Female Wheezers	5.0	0.8	1.5	4.6
Female Remissions	1.5	1.0	-	1.3

TABLE A.2.3
Average Spirometry values by site

	<u>BURNIE</u>			
	Male Wheezers	Male Remissions	Female Wheezers	Female Remissions
F.V.C.*	1.54	1.40	1.28	1.61
F.E.V. $\frac{1}{2}$ **	0.97	1.05	0.71	1.11

	<u>DEVONPORT</u>			
F.V.C.	1.84	1.96	1.30	1.65
F.E.V. $\frac{1}{2}$	1.09	1.52	0.65	1.20

	<u>MIDLANDS</u>			
F.V.C.	1.62	1.80	1.56	-
F.E.V. $\frac{1}{2}$	1.15	1.25	1.20	-

	<u>HOBART</u>			
F.V.C.	1.16	2.13	1.48	1.75
F.E.V. $\frac{1}{2}$	0.69	1.41	0.86	1.15

* F.V.C. Forced Vital Capacity (litres)

** F.E.V. $\frac{1}{2}$ Forced Expiratory Volume in half a second (litres)

TABLE A.2.4

The relative use of drugs for asthma by geographic area and total sample

	Burnie	Devonport	Midlands	Hobart	Total
SYMPATHOMIMETICS					
Ventolin (salbutamol)	15	36	60	32	32
Alupent (orciprenaline)	-	14	-	-	3
Actifed (pseudoephedrine)	-	14	-	-	3
Sudafed (")	-	4.5	-	-	1
Demazin (")	5	-	-	-	1
Polaramine (phenylephrine)	-	-	40	5	6.5
Bricanyl (terbutaline)	-	-	-	2.5	1
XANTHINES					
Quibron (theophylline)	-	4.5	-	2.5	2
Nuelin (")	10	23	10	2.5	10
Tedral (")	-	4.5	-	2.5	2
Elixophylline (theophylline)	7	-	-	-	3
Brondecon (Choline theophyllinate)	30	9	60	49	37
STEROIDS					
Prednisolone (prednisolone)	-	9	-	7	5
Becotide (beclamethasone)	-	9	10	5	5
ANTI-HISTAMINES					
Vallergan	-	4.5	-	-	1
Tacaryl	-	4.5	-	-	1
ANTIBIOTICS					
	15	9	30	10	13
INTAL (Sodium cromoglycate)	-	14	-	34	18
NON-PRESCRIPTION ITEMS					
	10	-	20	2.5	5

N.B. Numbers represent the percentage of the sample using a particular drug. Totals do not sum to 100 because of multiple usage.

TABLE A.2.5

The percentage of wheezers in each area who associated their attacks with environmental phenomena

	BURNIE	DEVONPORT	MIDLANDS	HOBART
Sea air/sea breeze	5	4.5	-	15
Cold air	25	14	-	22
Windy weather	5	14	-	10
Easterlies	25	32	-	-
Fog	-	11	30	2
Smoke	30	-	-	-
Snow	-	-	-	12
Weather Changes	65	36	50	17
Trips to the Beach	45	14	-	41

N.B. The nature of these associations was not prompted; the respondents were merely asked to name any "triggers" for their attacks.

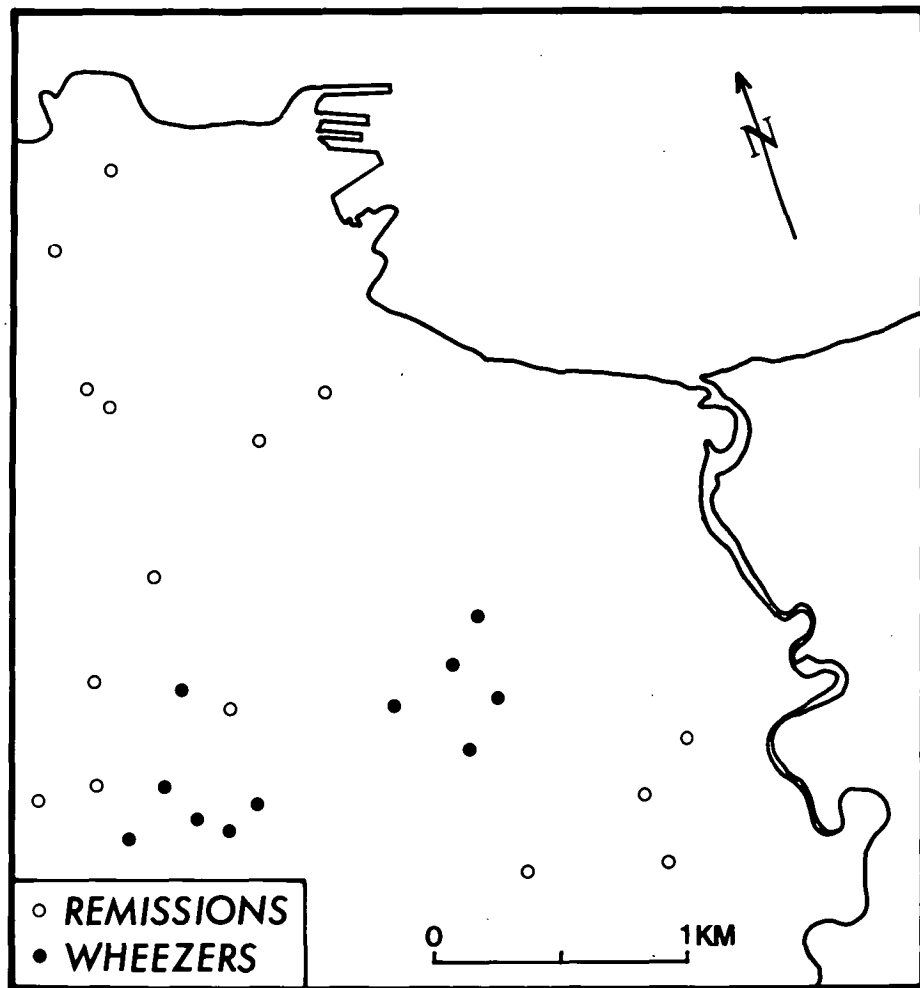


Figure A.2.1 Residential Location of Asthmatics from the Burnie Sample.

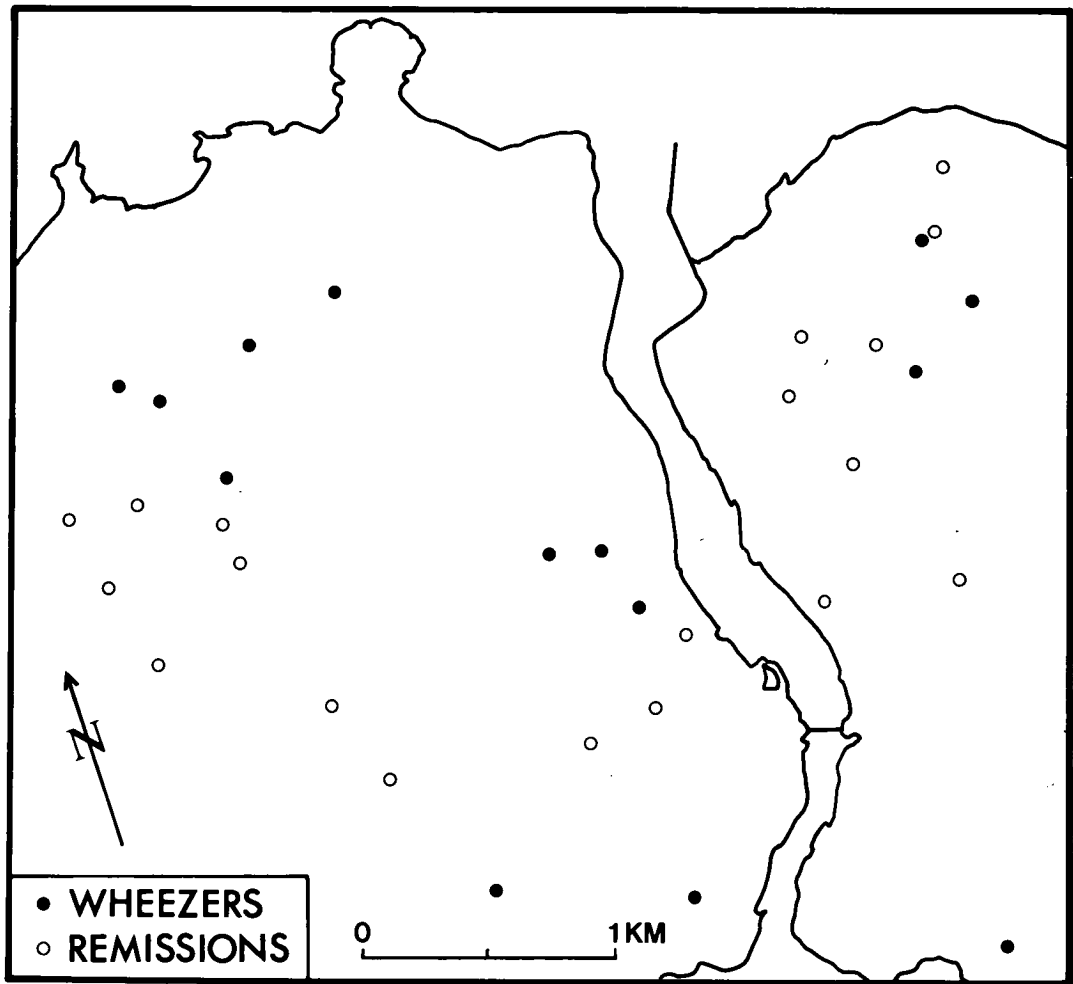


Figure A.2.2 Residential Locations of Asthmatics from the Devonport Sample.

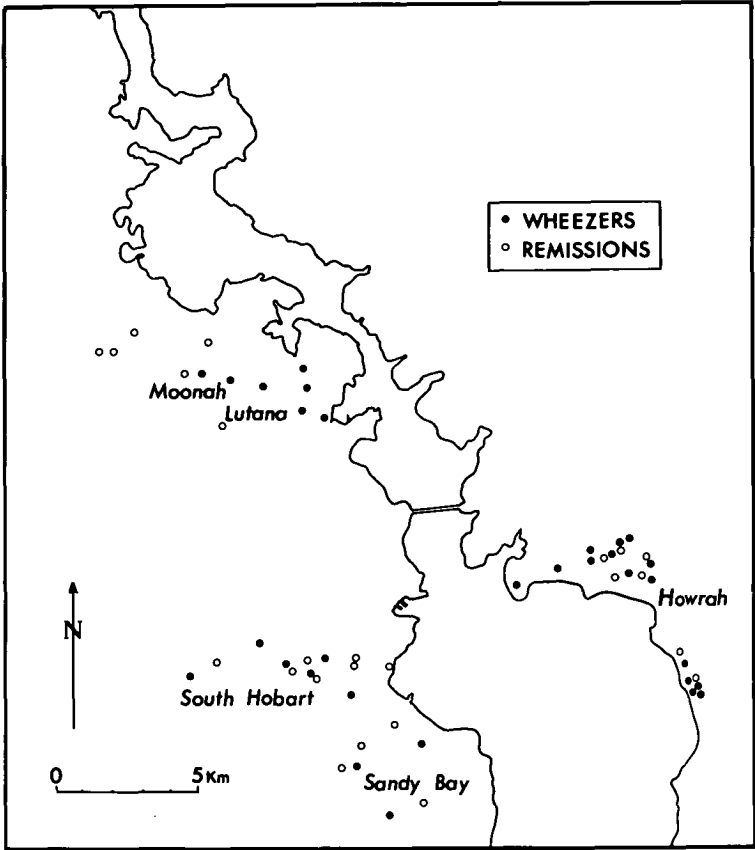


Figure A.2.3 Residential Location of Asthmatics from the Hobart Samples.

Name	Address	Year 1977	1978
Month J F M A M J J A S O N D	(circle the right one)		

DAY	WHEEZING	COUGHING	SNEEZING	OTHER COMMENTS
1				
2				
3				
4				
5				
6				
7				
8				
9				
10				
11				
12				
13				
14				
15				
16				
17				
18				
19				
20				
21				
22				
23				
24				
25				
26				
27				
28				
29				
30				
31				

If no complaint on any day just leave the space blank or draw a line through it. When there is an attack note the approximate time it started, approximately how many hours it lasted, and judge how severe it was on the following scale:

- 1 wheezing
2 slightly breathless
3 breathless
4 very breathless

If any medication is used e.g. Intal make a note in the last column.

Only make a note if the child makes more than half a dozen coughs during the day. Note the time of day that they are worse e.g. morning, afternoon or evening. If possible, try to record whether the cough is dry or loose.

Record the number of sneezing attacks experienced during the day.
If they are worse at any one time e.g. morning, afternoon etc., please
make a note.

This space is provided to make comments about drugs used to control attacks. It is also useful for you to record any impressions you might have about possible causes that may have brought the attack about: for example, dust, excitement, emotional upset, exercise, cold air, cold in the head, hayfever, the weather and unusual foods might all be entered in this column, as might any other items.

APPENDIX THREE

THE MEASUREMENT OF ATMOSPHERIC POLLUTION (ACID-GASES) IN HOBART

Introduction

Although it was suspected that pollution levels in Hobart would be low compared to other capital cities in Australia and insignificant when compared to the industrial giants of Europe and North America it was, nevertheless, decided to measure some of its aspects in order to examine its relationships to respiratory morbidity (wheeziness). This has been a controversial issue for many years. The effects of acute and/or chronic exposure to particulate and/or gaseous pollutants have continued to form the basis of debate by experts from several disciplines and countries.

The measurement of pollution in Hobart was restricted to the collection of 24-hour, integrated dose-levels of acid-gases. This limitation was enforced by the type of available equipment for pollution monitoring. The Department of the Environment kindly lent three acid-gas samplers for the duration of the study. These samplers operated sequentially for eight days and used the acidimetric hydrogen peroxide method.¹

Method of Measurement

In the acidimetric hydrogen peroxide method air is pumped continuously through a tube that leads from the sampling point to the apparatus. The apparatus consists of eight sets of filter clamps, eight Dreschel bottles, a gas meter and a suction pump (see figure A.3.1). An eight port valve changes the flow of air from the first filter clamp-Dreschel bottle assembly to the second after 24-hours has elapsed and so on.

On the eighth day of sampling the first seven filter papers and bottles are replaced.

The air is first drawn through a filter paper to remove any particulates from it. In the Dreschel bottle the filtered air is bubbled through a dilute solution (1 vol.) of hydrogen peroxide that has been buffered to a p.H. of 4.5 to prevent the formation of carbonic acid from ambient carbon dioxide. Acid gases, particularly sulphur and nitrogen oxides, are converted to sulphuric and nitric acids respectively at this stage.

In the laboratory the exposed samples of hydrogen peroxide are titrated against N/250 sodium borate solution using B.D.H. "4.5" indicator. The amount of acid-gases in the air sampled is estimated by the following formula:

$$C = \frac{4520T}{V} \text{ ug/m}^3$$

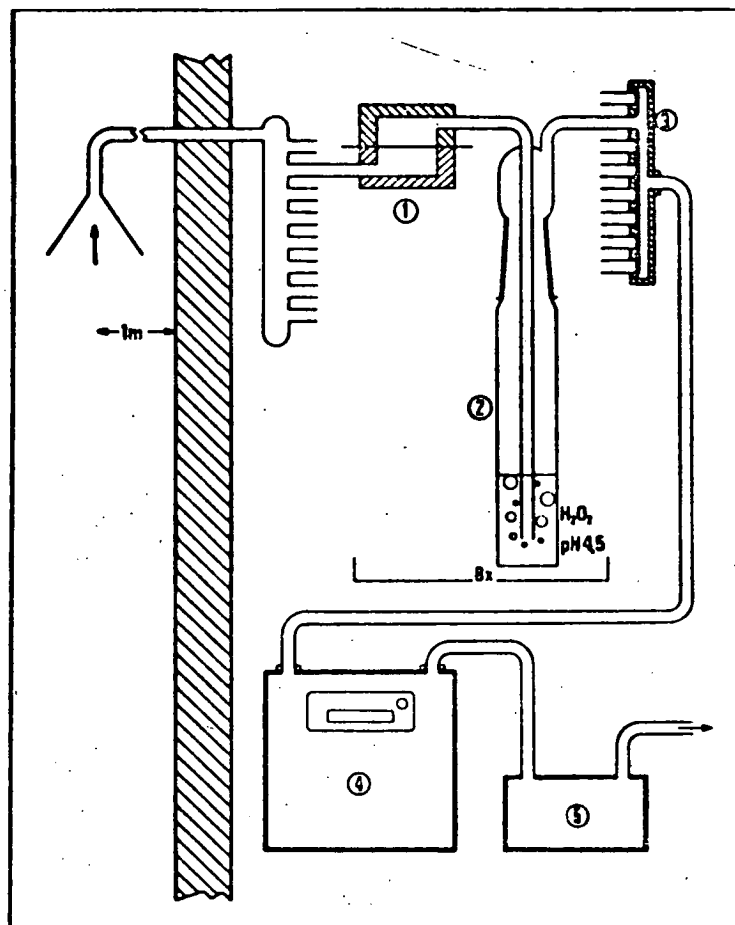
where, C is the concentration of acid-gases in microgrammes per cubic metre

T is the amount of N/250 sodium borate required to neutralise the peroxide solution in mls., and

V is the volume of air in cubic feet obtained from the gas meter readings.

Temporal Variation

Because of the small number of samplers it was impossible to establish a dense network of continually monitored sites. The machines used were known to be prone to breakdown, particularly the eight-port valve mechanism, and this necessitated holding



1. Filter Clamp
2. Peroxide Bubbler (Dreschel Bottle)
3. Valve-Port Assembly
4. Flow Meter
5. Pump

Figure A.3.1 Schematic Diagram of Acid-Gas Sampler

one machine in reserve at all times. At the start of the project two objectives were considered to be important:

a) the establishment of a baseline station to be continuously monitored for eighteen months in order to obtain a time series of daily values, and

b) the investigation of the spatial variability of daily acid-gas levels within the urban area of Hobart.

To satisfy the first objective a site had to be chosen that would represent an average urban exposure level. Private residences were excluded from consideration because of the high nuisance value involved in housing the machinery over an eighteen month period. Many public buildings were excluded because of the security risk involved in mounting the input tube, external to, and projecting one metre, from a window two metres above street level. An ideal site was eventually found in the ground floor offices of the old Lands Department building in Davey Street, Hobart. The kind permission of the occupants, the Tasmanian Council of Social Services and the Public Buildings Committee was readily obtained. Here the inlet tube was protected from interference by pedestrians by a two metre wide "moat" that gave light and some sort of ventilation to the basement windows. This "moat" was fenced in by iron railings. The combination of "moat" and railings rendered the tube inaccessible to all but the most determined and athletic vandal.

From this site the temporal variability of acid-gas levels was measured on a daily basis between the 27th August 1977 and the 3rd June 1978 with a gap in January 1978 due to a

machinery breakdown. Daily levels ranged from 0 to 45 ug/m^3 . These exposures were well within the acceptable standards.² For the 250 days of record the mean value was 21 ug/m^3 this compared favourably with the annual mean daily standard of 60 ug/m^3 .

The entire series of daily values is recorded in table A.3.1. When these data are averaged by day of the week the means differ little from the overall daily mean. Sunday's average is low at 19.1 ug/m^3 and Thursday's average is high at 22.2 ug/m^3 but neither of these is statistically significant. This indicates the lack of affect by the working week upon pollution levels. Some difference is seen, however, when monthly daily means are compared. These are given at the foot of the columns in table A.3.1 and they range from 28.1 ug/m^3 in March to 10 ug/m^3 in June. The Summer months' daily means were twice the average and the Winter months' means were half of the average value.

This seasonal distribution was thought to depend upon meteorological factors. Rainfall, for example, would have scoured acid-gases from the air; bright sunny days would have encouraged their persistence and build-up, and wind would have had dispersed them. A multiple regression analysis was conducted to see how much of the variation in acid-gas levels could be explained by weather factors. The best regression equation was obtained using five variables; 3 p.m. wet-bulb temperature, diurnal range, sunshine hours, 9 a.m. humidity and evaporation.

TABLE A.3.1

Acid-gas levels in Davey Street, Hobart 1977-78

DAY	AUG	SEP	OCT	NOV	DEC	JAN	FEB	MAR	APR	MAY
1		14	11	11	33	-	37	17	23	20
2		19	11	11	30	-	42	28	25	31
3		22	15	8	25	-	31	23	31	17
4		17	14	4	24	-	28	20	20	31
5		10	10	14	28	-	28	28	20	40
6		14	20	14	30	-	31	25	11	34
7		14	13	14	34	-	25	34	11	23
8		15	12	14	23	-	31	34	11	28
9		15	15	15	26	-	20	34	9	28
10		10	19	16	31	-	31	28	17	28
11		22	24	8	22	-	38	28	11	20
12		20	24	8	30	-	28	28	11	14
13		12	21	8	34	-	28	31	14	23
14		17	21	7	33	-	31	34	17	14
15		19	14	7	30	-	34	40	9	23
16		26	13	12	25	-	23	37	9	20
17		21	12	16	19	-	25	34	11	23
18		7	21	23	24	-	20	23	11	23
19		10	21	32	28	-	20	37	11	17
20		9	15	33	30	-	31	31	23	14
21		9	16	31	39	-	31	45	23	23
22		9	8	32	33	-	14	23	20	25
23		10	4	36	28	-	14	31	17	25
24		11	18	42	28	-	20	23	14	11
25		10	28	39	33	-	17	25	11	9
26		15	28	33	28	-	20	23	20	9
27	25	19	28	32	22	-	25	20	31	9
28	14	21	30	31	20	-	11	23	17	7
29	13	18	33	23	18	-	11	23	14	0
30	7	24	8	26	22	-	-	23	20	7
31	3	-	3	-	26	-	-	17	-	9
MEAN	12.4	15.3	17.1	20	27.6	-	26.2	28.1	16.4	19.5

The equation was as follows:

$$\begin{aligned} \text{Acid gas level} = & 0.31 + 0.82 \text{ 3 p.m. WET BULB} \\ & \text{TEMPERATURE} - 0.53 \text{ RANGE} + 0.62 \text{ SUNSHINE} + 0.15 \\ & 9 \text{ a.m. RELATIVE HUMIDITY} + 0.83 \text{ EVAPORATION} \end{aligned}$$

Actually, this regression was not very successful. Its r^2 value was only 11.6 per cent and the standard error of the estimate was 9.8. The variables included in the equation, however, suggest the role of Summer anticyclonic inversion conditions causing concentration of pollutants at certain times, for example, high afternoon wet-bulb, low temperature range, high sunshine hours, high morning humidity and high evaporation could all describe this sort of synoptic occurrence.

It is interesting to compare the Hobart values with some data collected by the Department of the Environment in Burnie during the Summer of 1973-74 (see Table A.3.2). The averages given are the daily means averaged over a week. These average values probably conceal a wide range in variation throughout the period of a week. Of the three sampling sites, Montello, a hilltop suburb to the West of the Australian Pulp and Paper Mills factories, gains the highest readings, (see figure A.3.2.). This site is frequently subject to fumigation by factory effluent. Emu Heights and Wivenhoe, to the South and East of A.P.P.M. respectively, are both much lower in exposure to this source. The probable origin of acid-gases is from the use of high-sulphur oil used in the factories' steam boilers.

There does appear to be some cause for concern in regard to the Burnie acid-gas levels. The average daily values for the

TABLE A.3.2
 Acid-Gas Levels in Burnie
 Weekly Averages ug/m³

	<u>Emu Heights</u>	<u>Wivenhoe</u>	<u>Montello</u>
<u>November 1973</u>			
Week 3	25	-	31
Week 4	8	45	86
<u>December 1973</u>			
Week 1	43	23	53
Week 2	78	57	73
Week 3	51	40	87
<u>February 1974</u>			
Week 4	40	49	113
<u>March 1974</u>			
Week 1	84	50	128
Week 2	72	69	78
Week 3	26	27	45
Week 4	55	46	100
<u>April 1974</u>			
Week 1	23	33	50

Source: Department of the Environment

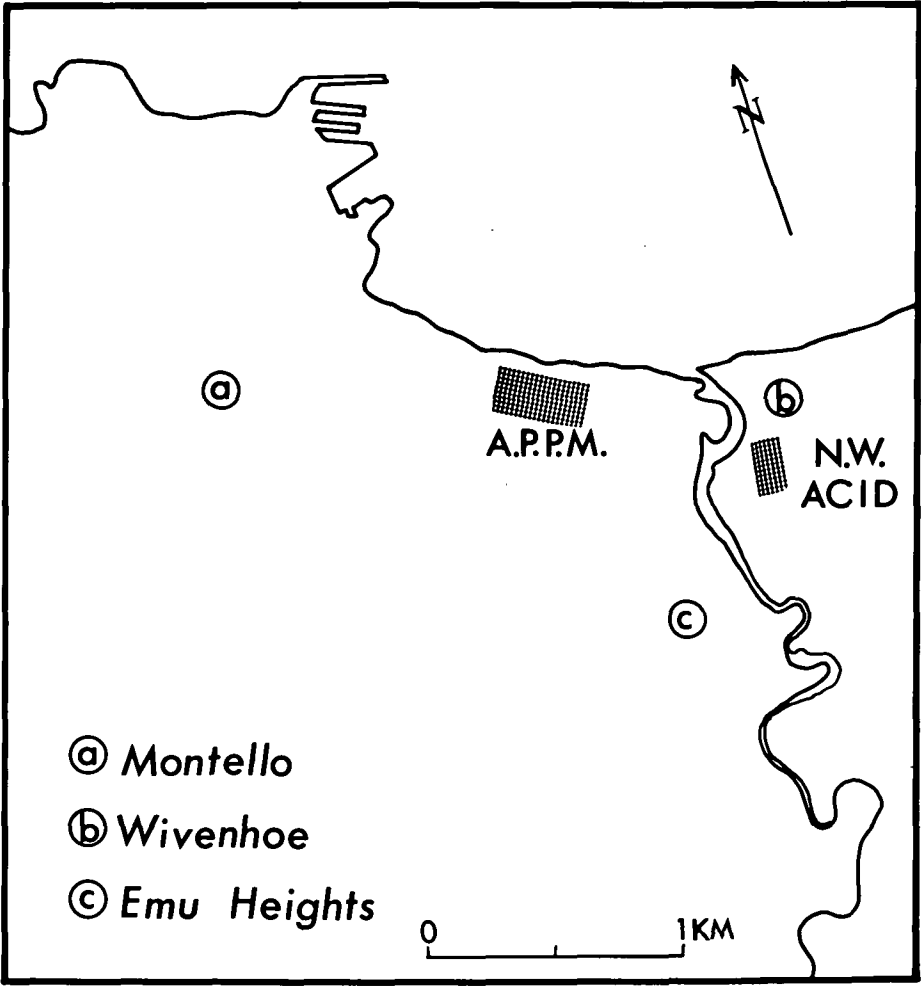


Figure A.3.2 The Location of Acid-Gas Sampling Sites in Burnie in Relation to Major Sources of Acid-Gases.

fourth week of February and the first and fourth weeks of March 1974 are all at least 100 ug/m^3 . This is well in excess of the standard annual daily exposure of 60 ug/m^3 . For the entire month of March 1974 Burnie's (Montello) average daily value is calculated at 88 ug/m^3 this compares with Hobart's level in March 1978 at 28 ug/m^3 . During the first week of March in Montello the average daily reading of 128 ug/m^3 may have been exceeded on individual days by as much as fifty per cent. The exacerbation of these acid-gas levels by anticyclonic inversions and sea breezes can be viewed as a tangible threat to health.

Spatial Variation

As was noted from the Burnie data, in addition to variation in levels over time was a variation in levels across space. The measured levels at Davey Street, Hobart, although representing the values of a representative urban location, did not necessarily describe the levels found in another part of town. Obviously, it was desirable to estimate how acid-gas levels varied relatively from place to place within the urban fabric. Ideally this should have been accomplished by using many samplers stationed in a dense network throughout the city. This would have resulted in a map or maps of simultaneous absolute values at the sampling points. Such a map could have served as a base for isopleths representing equal acid-gas levels over the city.

With only two or three samplers available a different strategy had to be adopted. As the Davey Street station was to

be the baseline monitor and was fixed in space, it was decided to move the other two samplers from site to site and to consider spatial variation on the basis of the ratios between the Davey Street values and the mobile sites' values. Seven days of measurements in a given location were summed and averaged and this was repeated for the synchronous Davey Street measurement. The mobile station's average divided by Davey Street's average and multiplied by one hundred thus expressed the mobile site's measurements as a percentage of the fixed site's value for the same time period. Seven day averaging also reduced any chance variation in individual daily values and any weekly cycles in acid-gas levels found in different locations around the city.

These ratios were mapped at points on a Hobart base map and then isopleth lines were drawn connecting points and interpolations of equal value. An isoline with the value of one hundred described, therefore, a domain with equivalent acid-gas levels to the base station in Davey Street. An isoline with a value of five hundred similarly represented levels of acid-gases five times the levels met at the Davey Street site.

Figure A.3.3 is an isopleth map of these ratios for the seven day averages. This map shows that, on average, the northern suburbs of Hobart on both shores of the Derwent have twice the acid-gas levels of the base station and the more southerly suburbs of South Hobart, Sandy Bay and Trammere.

The highest levels are observed in the Eastern shore suburbs of Geilston Bay and Lindisfarne where the ratio to the

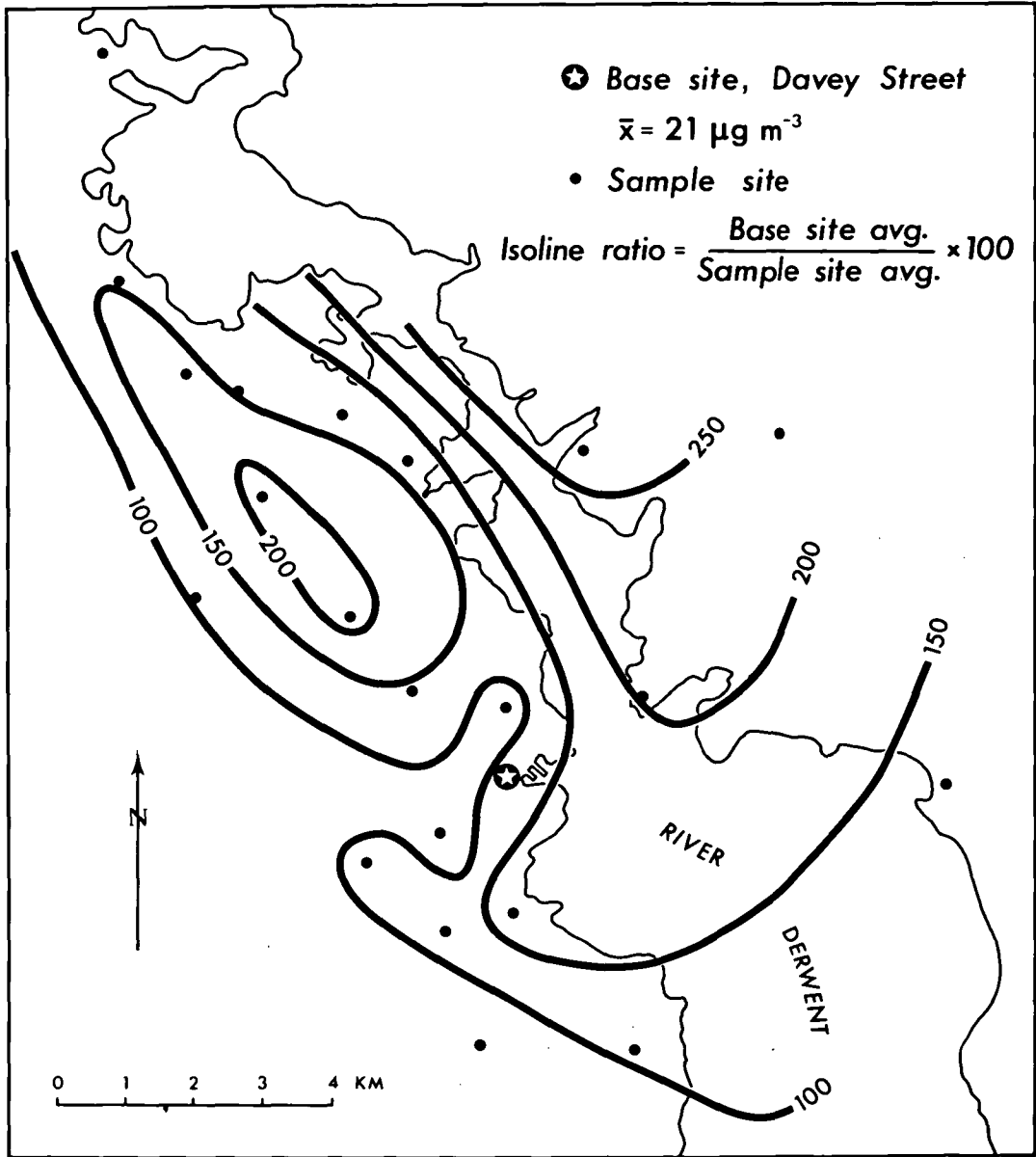


Figure A.3.3 Spatial Variation in the Average Acid-Gas Levels in Hobart.

base station is 250 and higher; 262 in Derwent Avenue, Geilston Bay. The source of acid-gases and other pollutants in this premium residential neighbourhood is from across the river. The average wind flow is from the Northwest channelled by the Derwent's estuary. Opposite and to the Northwest lies the largest zinc smelter in the Southern Hemisphere, Electrolytic Zinc (E.Z.). The levels downwind from this industry are higher than in the industrial park area of North Hobart and there is, therefore, no confusion about the source that is culpable. The average level in Davey Street is 21 ug/m^3 . The average level in Geilston Bay is therefore $21 \times 2.5 = 53 \text{ ug/m}^3$.

The average spatial variation in figure A.3.3 disguises the vast variation from day to day. Acid gases are mobile and extremely soluble in water. Day to day variations in wind direction, wind speed humidity and rainfall can affect acid-gas levels. During the study period, excluding rain days, three wind conditions were available for each site; light and variable winds, Northwesterly winds and Southwesterly winds. Figures A.3.4, A.3.5 and A.3.6 illustrate the spatial variations in acid-gas levels under these conditions (light, NW and SW respectively). Under conditions of little wind the acid-gas levels form an hour-glass shaped distribution with roughly circular concentrations on both sides of the river (figure A.3.4). The two high spots are, again, on the northern East-shore opposite E.Z. and in the industrial park suburbs on the northern West-shore suburbs. In both locations the levels are over 2.5 times the levels at the base station and in the Southern parts of the city (25 ug/m^3).

One site in Moonah has a ratio of 295 representing a level of 74 ug/m^3 .

During the northwesterly wind regime, figure A.3.5, the isolines become elongated parallel to the river valley. Again, the northern suburbs are higher than their southern counterparts. The highest levels are found on the East-shore and are obviously downwind from the E.Z. The gaseous effluents from this source are dispersed downstream subjecting most of the Eastern-shore shoreside suburbs to acid-gas levels 2.5 times the base station's level (23 ug/m^3); about 58 ug/m^3 .

The most extreme differentials are obtained during southwesterly winds, figure A.3.6. Here, the average value at Davey Street is only 15 ug/m^3 . Three times this level is reached in the West-shore industrial park area, 45 ug/m^3 . In this situation, however, the effluent from E.Z. is not dispersed downstream but is advected almost straight across the river with minimal dilution. The levels in Derwent Avenue, Geilston Bay are 5.5 times the Davey Street value, 83 ug/m^3 . This is well above the standard annual daily mean permissible level. These readings begin to rival those from the most polluted parts of Burnie.

Summary

Although Hobart's acid-gas levels are fairly low in comparison to larger and/or more industrialised centres, certain sites under particular conditions do appear to be at risk of significant pollution. Levels vary from place to place within

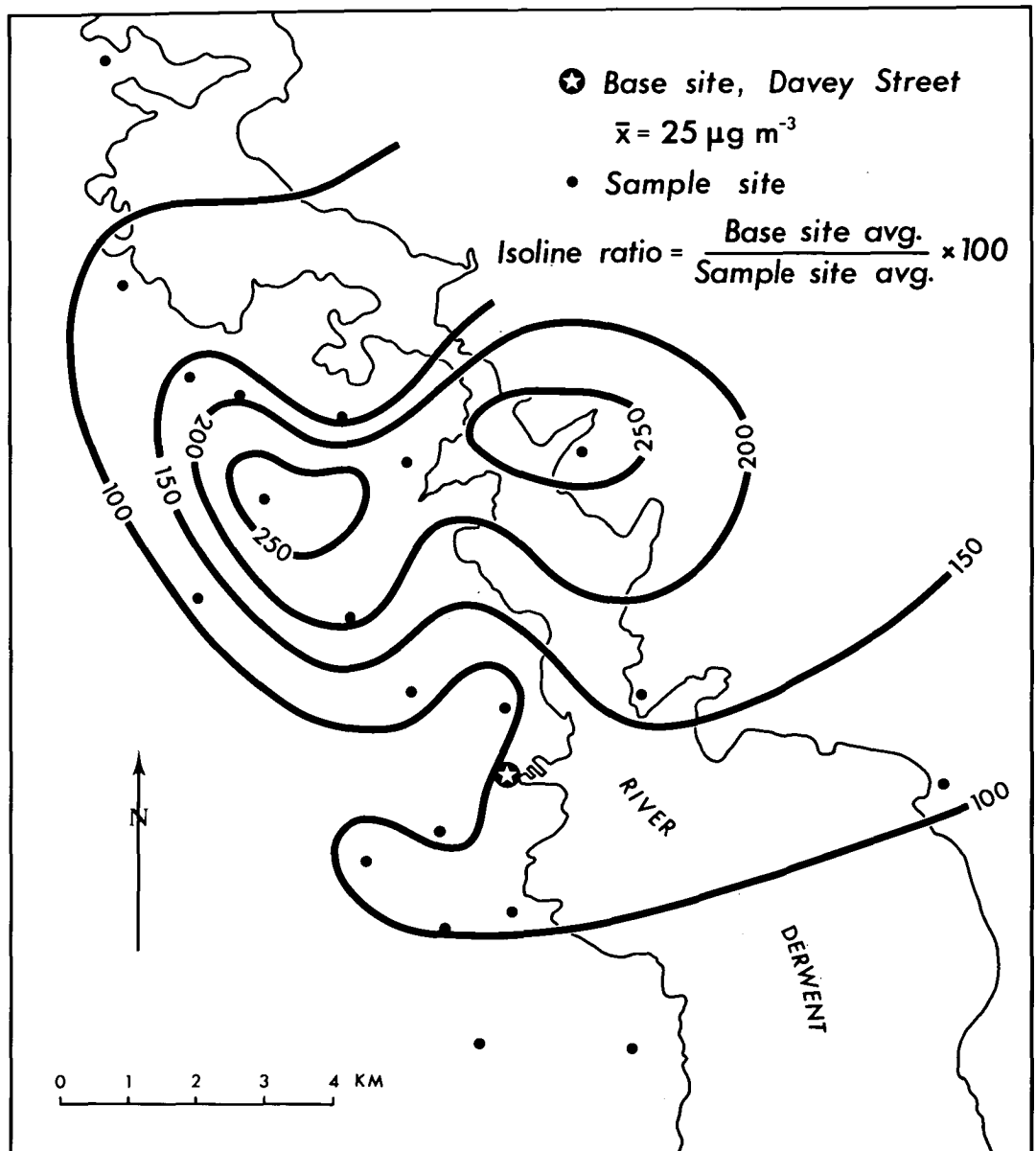


Figure A.3.4 Spatial Variation in Acid-Gas Levels in Hobart Under Conditions of Light and Variable Winds.

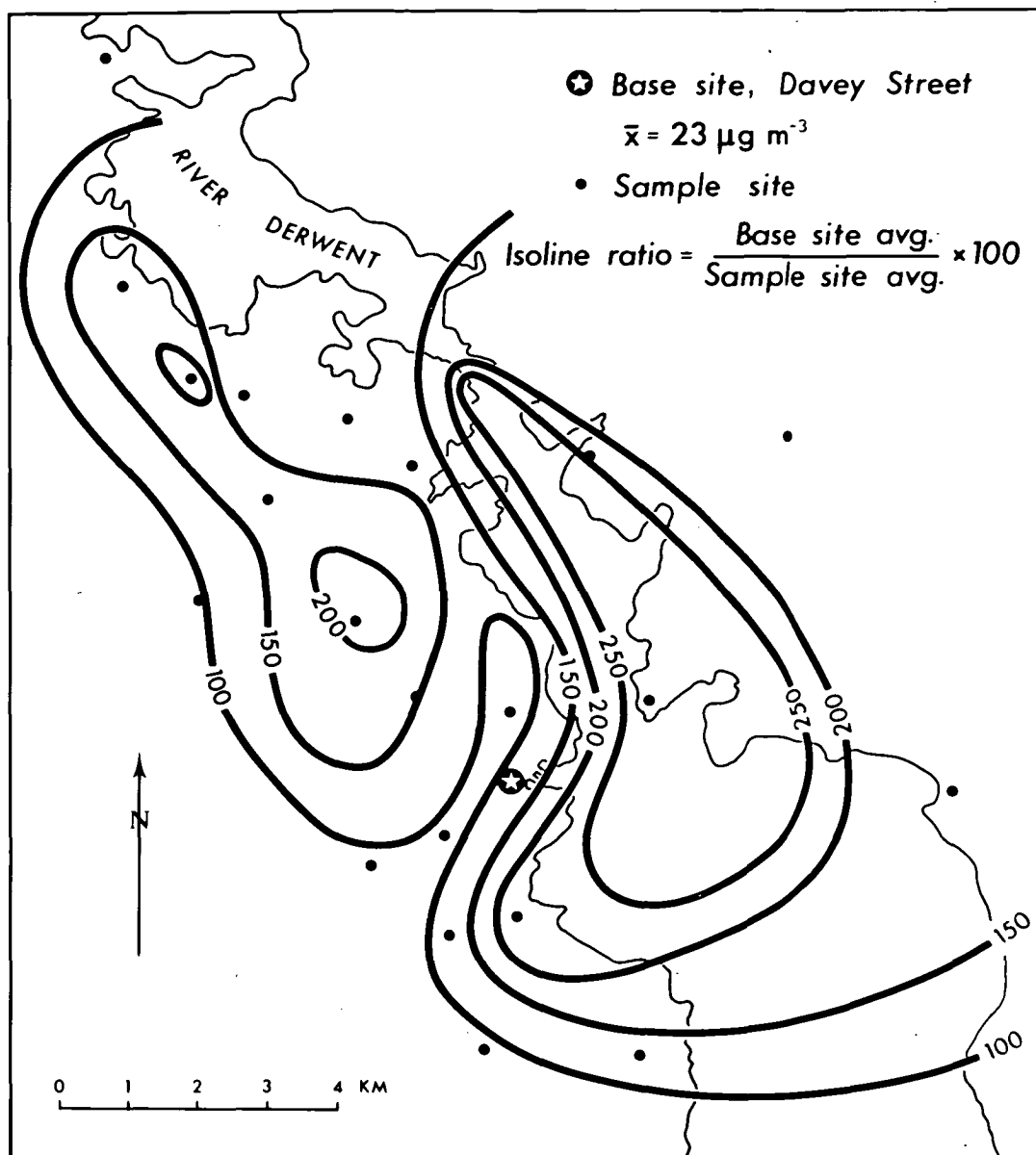


Figure A.3.5 Spatial Variation in Acid-Gas Levels in Hobart During a Northwesterly Wind Regime.

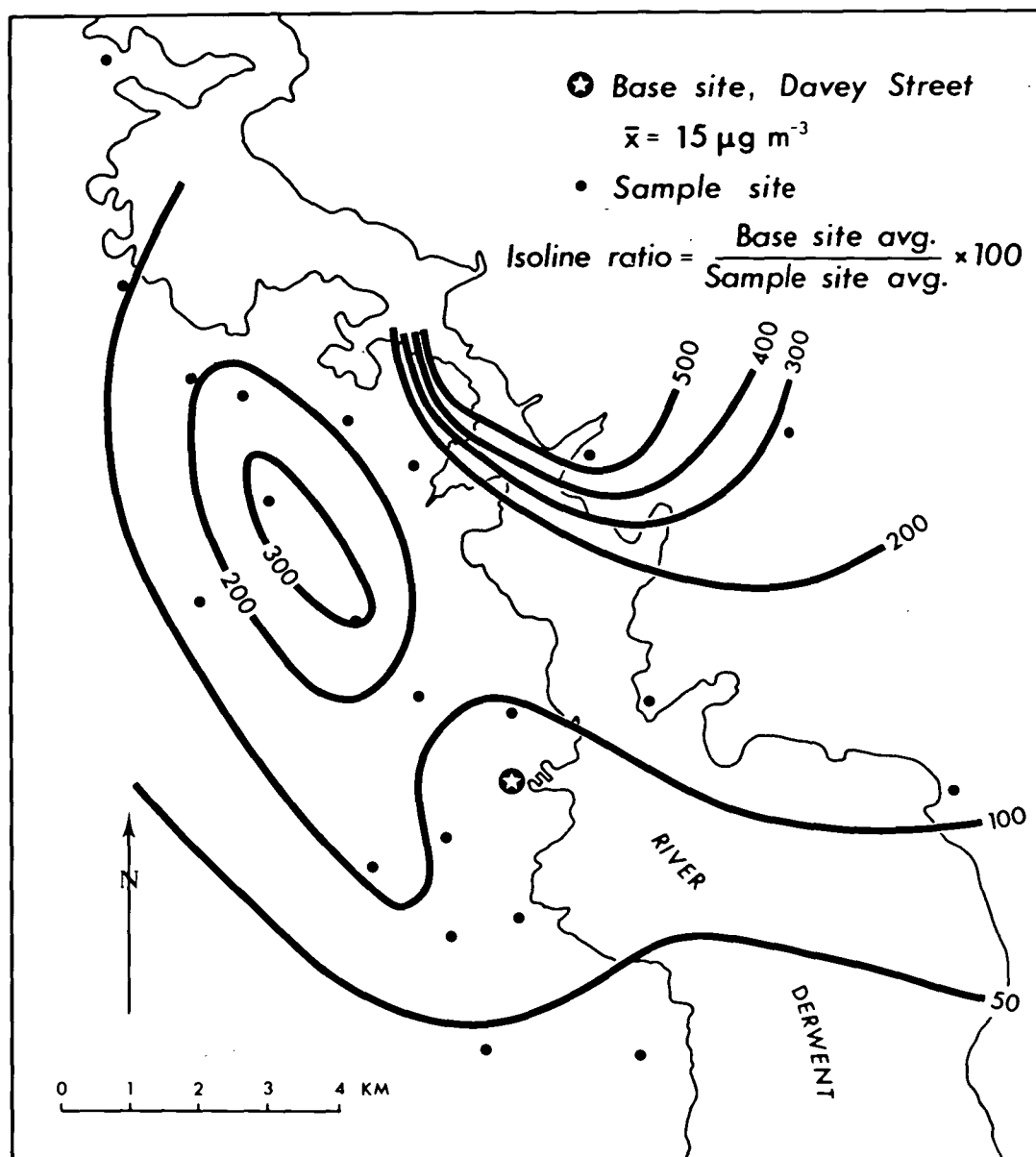


Figure A.3.6 Spatial Variation in Acid-Gas Levels in Hobart During a Southwesterly Wind Regime.

the urban fabric and are largely modified by wind direction. The most polluted area embraces the East-shore suburbs of Geilston Bay and Lindisfarne. These are downwind from the E.Z. a source of acid-gases particularly sulphur oxides. The next most polluted area combines Moonah, Lutana and Derwent Park on the West-shore. High levels of acid gases in this locality are probably due to the combined combustion of fuel oil from industrial, domestic and vehicular sources. South of the Tasman Bridge the levels are fairly consistent with those measured at the base station in Davey Street.

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APPENDIX FOUR

THE MEASUREMENT OF ATMOSPHERIC IONISATION

Apparatus and Method

The measurement of atmospheric ionisation was not a trivial problem. Indeed, the insulation and amplification requirements were so great as to cast doubt upon some of the previous attempts in this area.¹ In section one the subject of ionisation was reviewed briefly and the reader was referred to the work of Trevitt,² as an example of recent ionometer design. For a review of the physical principles of ionisation measurement the reader is again referred to this source. The sensor constructed for this research was based upon the prototype described there but was extensively modified with regard to insulation and circuitry.

Basically, the measurement system consisted of four components; a sensor, a power source, an amplifier and a recorder. The sensor consisted of a large cylindrical capacitor (see figure A.4.1); a Gerdien tube with a fan mounted on one end to produce a constant air flow through the device. The power source provided the bias voltage, the potential difference between the central electrode and the outer cylinder for different voltage and charge. It stepped the instrument through a sequence of voltages and charge signs that took 30 minutes to complete. In this way, a reading for every voltage-charge combination was taken twice in every hour. The sequence was: zero, +10V, zero, +100V, zero, +200V., zero, -10V, zero, - 100V, zero and -200V. These values represent the charge that was given to the central electrode; the cylinder was earthed. The different

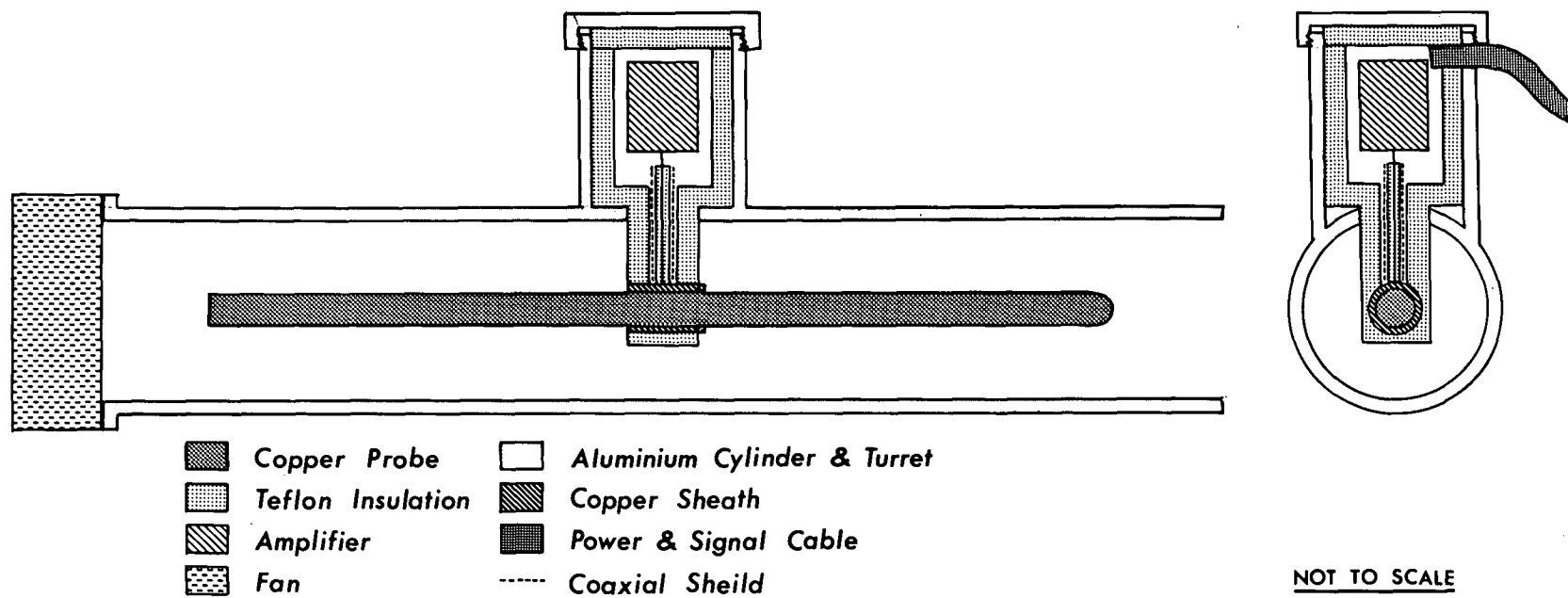


Figure A.4.1 The Gerdien Sensor

voltages collected different proportions of the population of ions that transitted the tube. Higher voltages, as well as collecting the small highly-mobile ions, collected medium and large ions also. A manual override was provided so that the automatic sequencing could be stopped and a desired population could be exclusively monitored.

From figure A.4.1 it can be seen that the central electrode was both mounted and insulated at its mid point by a structure that passed through the outer cylinder into a turret-like projection. This insulator was machined from a single piece of TEFLON to fulfil four functions: i) mechanical support of the central electrode; ii) electrical insulation of the central electrode, iii) electrical insulation of the "signal" from the central electrode, and iv) electrical insulation of the amplifier. The rod of TEFLON that projected into the cylinder was cross-bored and lined with a piece of copper tubing. The central electrode was sheathed in this thus making a good electrical contact. The supporting rod was hollowed out to accommodate a piece of TEFLON-insulated coaxial cable with a very snug fit. The central wire from this cable was soldered to the copper sheath by poking it through a pre-drilled hole in the sheath and then soldering it in situ and filing it smooth. The co-axial cable fed into the larger part of the TEFLON that was housed in the turret. Here the TEFLON had been hollowed out to a much larger extent enabling it to house the amplifier circuit (figure A.4.2).

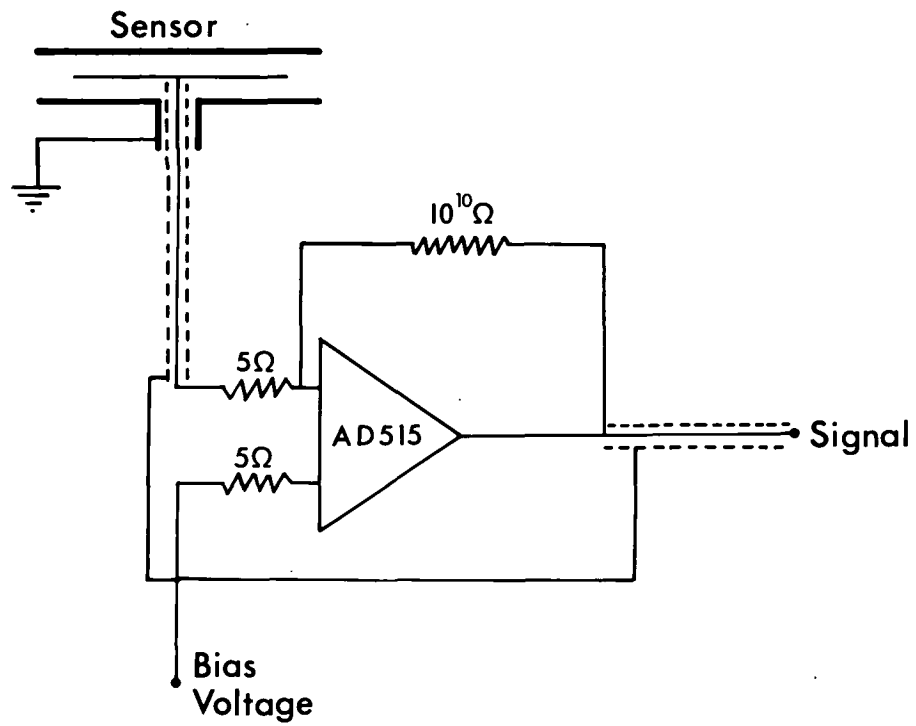


Figure A.4.2 Schematic of Sensor and Amplifier Circuit Diagram

The flow of ions to the central electrode produced a tiny current that flowed from the electrode to the amplifier. The current expected from theoretical calculations was minute, of the order of 10^{-13} amps, hence the strict attention to insulation. The shielding on the co-axial cable that carried the signal was held at the bias voltage (the voltage given to the central electrode) to prevent any possible dielectric effects during its passage through the outer cylinder. The use of the turret to house the amplifier was advantageous because it enabled the distance that the "signal" had to travel to be minimised and it provided an increased efficiency of high-quality insulation. A small sack of silica gel was eventually included within the turret to scavenge any traces of moisture.

The next problem was the amplification of the signal to a level that could be recorded by commercially available machines without introducing significant amounts of noise. The usual method of measurement had been to use a commercial electrometer but their expense precluded their use in this study. Instead, the low-level current to voltage converter used by Trevitt was modified to reduce noise by a factor of ten. This circuit included a F.E.T. (field effect transistor) input - operational amplifier; Analog Devices AD515 (see figure A.4.2.). This offered the lowest input bias currents available (50 fA). This op-amp acted in concert with a feedback resistor of 10^{10} ohms. This configuration converted a current of 10^{-13} amps into 10^{-3} volts. A signal of this magnitude was readily input to conventional chart recorders after smoothing by a simple RC filter.

During test runs it was noticed that the sensor was affected by electrical events inside the building (for example, lighting and power switches) and also by the movements of charged bodies (for example, human beings, particularly those dressed in synthetic fabrics). The machine was, therefore, housed in an earthed metal case in which the sensor stood upright. Openings beneath the top lid and at the bottom of the case were screened with wire mesh and allowed the free entry and exhaust of air. This form of shielding greatly reduced the extraneous effects.

Figure A.4.3 gives an example of chart recorder output. The signal in millivolts is recorded for each voltage-sign combination twice every hour. If desired, these voltages can be converted into ion numbers as follows:

$$\text{if } V = i R \text{ volts}$$

$$\text{then } i = \frac{V}{R} \text{ amps.}$$

If R equals 10^{10} ohms and one ampere is defined as one coulomb of charge per second and one coulomb is equivalent to 6.284×10^{18} electrons or protons, then the number of ions, assuming unit charge, equals

$$\frac{V}{10^{10}} \times 6.284 \times 10^{18} \text{ per volume of air sampled in one second}$$

A signal of one millivolt would be equivalent to:

$$6.284 \times 10^{18} \times 10^{-10} \times 10^{-3}$$

$$\text{or } 6.284 \times 10^5 \text{ ions/second.}$$

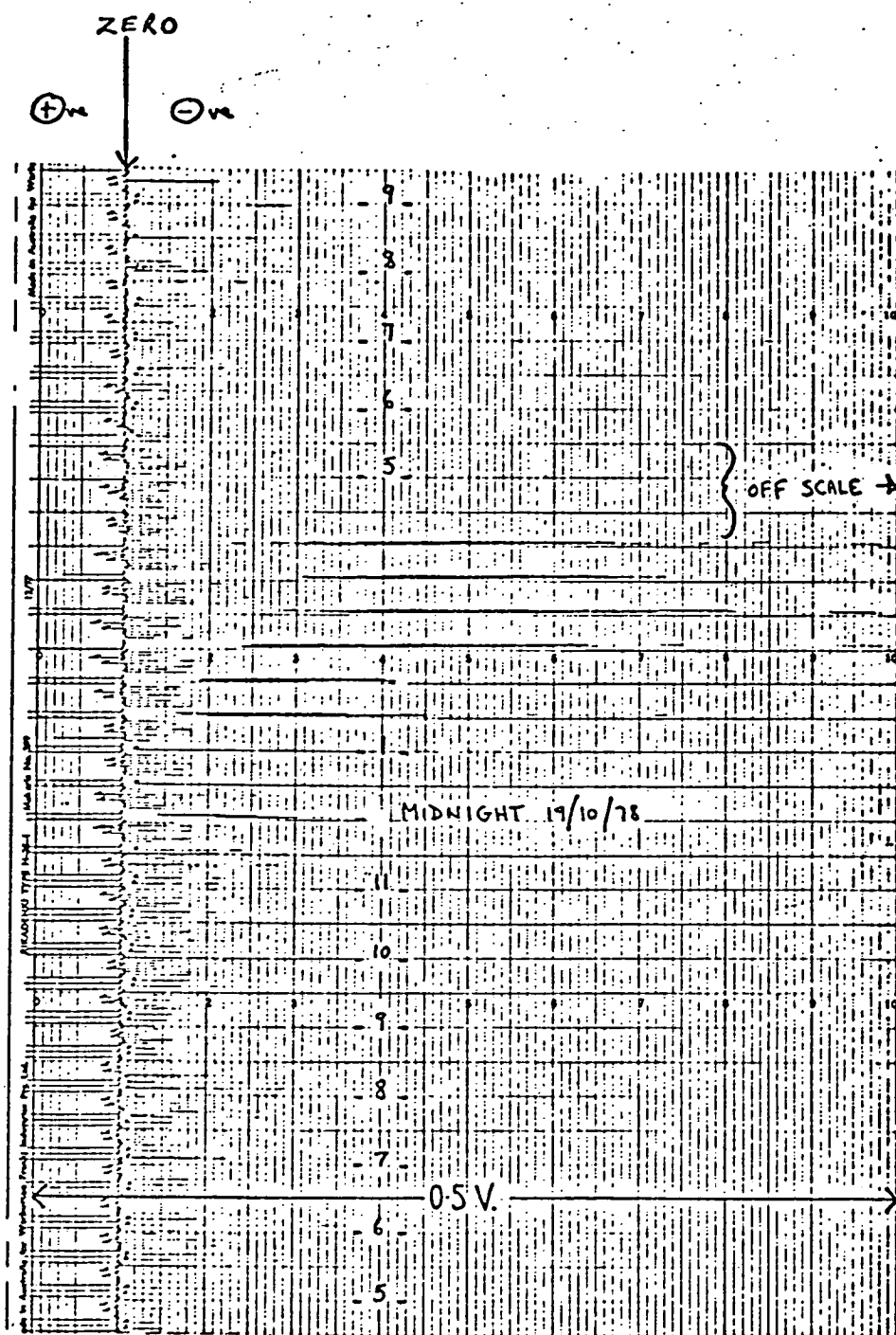


Figure A.4.3 Example of Chart Recorder Output

The flow rate through the Gerdien tube is $49550 \text{ cm}^3/\text{sec}$. A one millivolt signal is therefore

$$\frac{6.284 \times 10^5}{49550} \text{ ions/cm}^3 \text{ sec.}^{-1}$$

$$\text{or } 12.7 \text{ ions/cm}^3 \text{ sec.}^{-1}$$

The average number of ions in the air over open land is estimated at between 1000 and 2000 per cubic centimetre³. The presence of 2000 ions/cm³ would give a reading of 160mV.

Figure A.4.4 illustrates one day of recorded measurements. The millivolt signals are converted into ion densities per cubic centimetre on the vertical axis. These measurements were taken inside an office on the second floor of the Geography and Geology building at the University of Tasmania. The windows to this room were always open to allow the free movement of air from the outside. To assist interpretation, only the ion densities sampled at ten volts and 200 volts were illustrated. The ion densities sampled at 100 volts were about sixty per cent of the densities measured at 200 volts and they followed the same variations. At ten volts the ion densities were about fifteen per cent, on average, of the densities measured at 200 volts. From the graph, the maximum density of small positive ions was 36 ions/cm^3 . This was twice the amount of the small negative ions. At 200 volts the maximum positive ion density was 215 ions/cm^3 and the negative ion density 130 ions/cm^3 . The proportion of positive to negative ions was 5:3; slightly higher than the usual reported value of 5:4.⁴

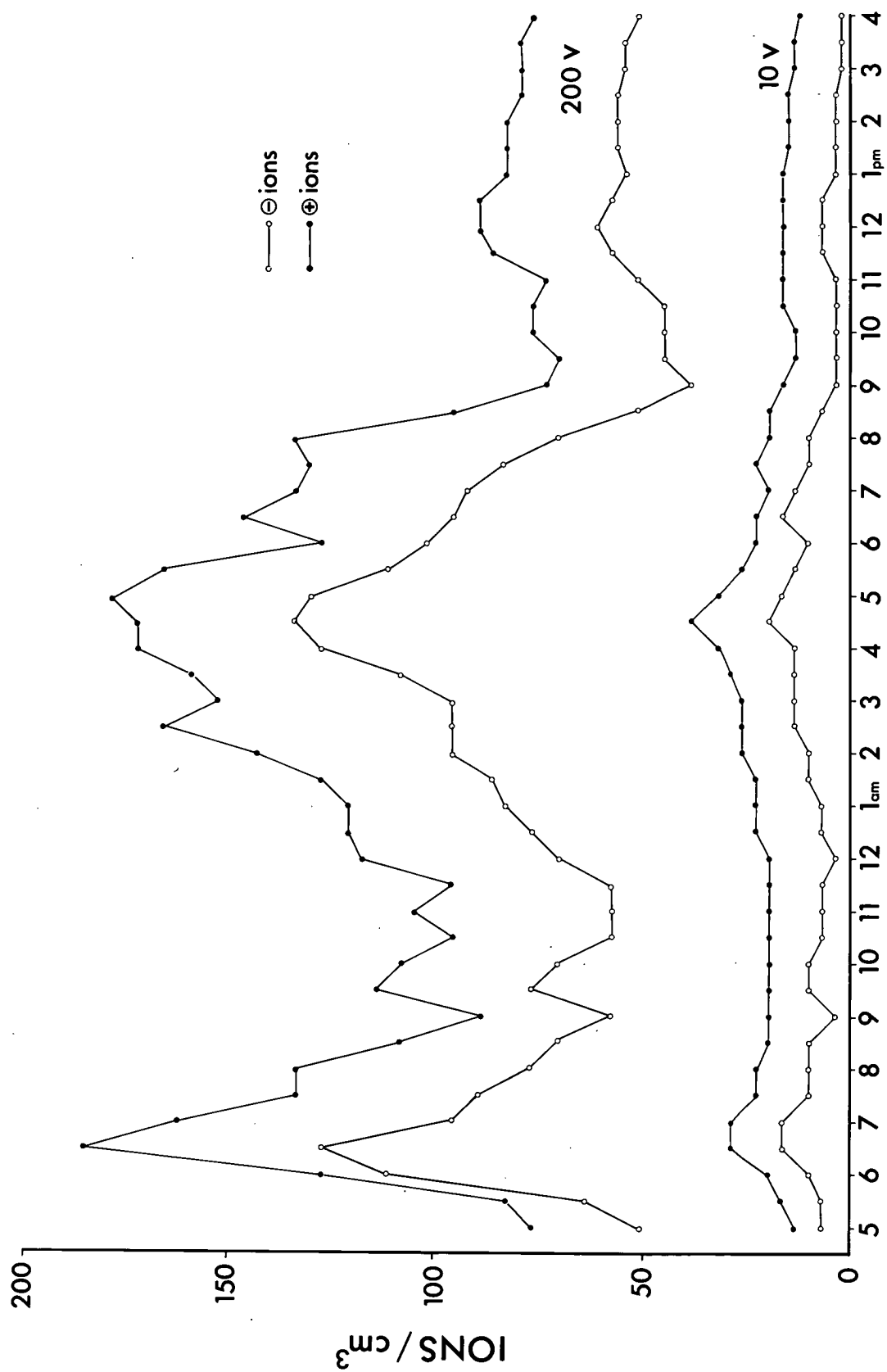


Figure A.4.4 The Diurnal Variation of Ion Densities in Hobart 26-27 June 1978

The graph in figure A.4.4. was typical of the diurnal variation of ions in Winter time Hobart. They demonstrated a peak between 2 a.m. and 6 a.m. with a maximum around 4.30 a.m. During the daylight hours the levels were usually lower; 30-50 per cent of the early morning maximum. These measurements were started in late June 1978 and the diurnal pattern described above remained consistent for just over two months. The only variability that was noticed was a slight increase during rainfall but this was not as significant as the diurnal range. Often, rainfall merely introduced more short-frequency noise into the signal, making it more spikey and blurred.

In September, however, something unusual began to happen; the early morning, negative-ion maximum increased by at least a thousandfold. Daytime readings maintained the same levels as during the Winter months but at about 3 a.m., on average, the signal suddenly went outside the measurable range of the recorder (2 volts), and remained off-scale until 7 a.m. or 8 a.m. This pattern soon became the norm and was only disturbed by strong weather when the Winter pattern was resumed. This new peak signal represented air that possessed an ionic density of at least $24,000 \text{ ions/cm}^3$, and probably far in excess of this. During this period the positive ion density only rose from about 180 ions/cm^3 to 250 ions/cm^3 .

Early morning observations at these times were able to detect changes in the direction and strength of the wind at the onset of these peak times. The enormous surplus of negative ions was associated with light winds from higher slopes inland

from the Coast. The low ion counts during Winter and in daytime were probably due to the combined affects of aerosols in sea and urban air absorbing the highly-mobile negative ions onto their surfaces. Land-air under night conditions in Spring contained both a large amount and a large proportion of negative ions. To examine this further, a recorder was situated at a rural farm site 60 kilometers inland from Hobart for three nights. In this location the daytime values were ten times the average coastal values and the proportion of negative ions to positive ions was 2:1. At night the signal went off-scale earlier (10 p.m.) and came back on scale later (9.30 a.m.). Measurements made in Burnie over the course of a weekend were very similar to Hobart's pattern.

If atmospheric ionisation levels were implicated in wheezy morbidity incidence then the strong fluctuations observed in the Spring should have been reflected in an increased or decreased number of episodes of wheezing. Comparing the months of August (1-28 only) and September 1978 this difference in incidence did not appear in the Hobart data. The relationship of ionisation to the prevalence and incidence of asthmatics, however, was a different question that would have required further prospective investigation. The "interior" group defined by the discriminant analysis from section two comes to mind in this regard. The heightened values of ionisation at inland locations contrasting with the low-ion environments in towns and the coast presented a good comparative basis for further research. This is in progress.

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